1994 Index to *Missouri Epidemiologist*

KEY

J/F94 = January/February 1994
M/A94 = March/April 1994
M/J94 = May/July 1994
A/O94 = August/October 1994
N/D94 = November/December 1994

Division of Environmental Health and Epidemiology Telephone and HOTLINE Numbers

Childhood Lead Poisoning Program	(800) 575-9267
Communicable Disease Consultation	(314) 751-6113
Communicable Disease Reporting	(800) 392-0272
Community Environmental Health	(314) 751-6095
Division of Environmental Health and Epidemiology	(314) 751-6080
Environmental Epidemiology	(800) 392-7245 (314) 751-6102
Immunization	(314) 751-6133
Occupational Fatality HOTLINE	(800) 392-7245
Office of Epidemiology	(314) 751-6477
Radiological Health	(314) 751-6160
Radon HOTLINE	(800) 669-7236
Sexually Transmitted Diseases/HIV	(800) 359-6259
Tuberculosis Control	(314) 751-6122
Vaccines For Children Program	(800) 219-3224
Veterinary Public Health	(314) 751-6136

Missouri EPIDEMIOLOGIST

Volume XVI, Number 1 *January-February 1994*

Laboratory Testing for E. Coli O157:H7 Prior to the First Year of Reporting in Missouri

Michael Fobbs

E. coli O157:H7 is a bacterium responsible for gastrointestinal illness with bloody diarrhea as a common symptom. Its importance can be demonstrated by a multistate outbreak of over 500 cases associated with a restaurant chain in the fall and spring of 1992-93. The largest waterborne outbreak reported in the literature to date, with over 243 cases of *E. coli O157:H7*, occurred in Cabool, Missouri in December 1989 - January 1990.

E. coli O157:H7 was made reportable in Missouri effective June 25, 1992. In the period from July 1992 through July 1993, 24 cases have been reported to the Departmentof Health's Bureau of Communicable Disease Control.

A survey was sent to 169 microbiology laboratories in the state to determine the background level of *E. coli O157:H7* in the twelve months before it became a reportable condition (07/01/91- 06/30/92).

Responses were received from 135 (79.9%) laboratories. A total of 27 (20.0%) laboratories stated that they tested for *E. coli O157*. Of these, 26 (96.3%) stated that they used Sorbitol-MacConkey (SMAC) agar plates to detect *E. coli O157*. The other laboratory did not describe their procedure in detail. A latex agglutination procedure was also used to confirm *E. coli O157* by 18 of the 27 laboratories (66.6%). During this same time period, 19 (70.4%) sent their *E. coli O157* isolates to the State Public Health Laboratory (SPHL) to test for the H7 antigen. The SPHL and other reference laboratories are the only ones capable of testing for the H7 antigen. The Centers for Disease Control and Prevention also receives *E. coli O157* isolates for H7 testing and less than 1 percent are some other antigen.

Of the 27 laboratories that tested for *E. coli O157* only 8 (29.6%) did routine testing of stool samples for it. At 12 (44.4%) laboratories testing was done if there was bloody diarrhea and at 17 laboratories (62.9%) it was done at the physician's request whether or not there was bloody diarrhea. One lab tested all stool samples from April through December, but only tested at the doctor's request during the months of January through March.

In the period from July 1991 through June 1992, the surveyed laboratories reported they had 30 specimens test positive for *E. coli O157*. This is consistent with the 24 cases reported from July 1992 through July 1993. While there have been no confirmed waterborne or foodborne outbreaks during these time periods, the significant number of cases reported should remind us to be continually observant of the potential for outbreaks within communities in the state.

New surveys to determine the current levels of laboratory testing for *E. coli O157* will be initiated in the near future now that baseline levels have been established. Any increase in the number of laboratories doing this type of testing will increase the effectiveness of disease surveillance and subsequent control measures for this bacteria by allowing us to determine the demographics of the disease and whether any geographic clustering is occurring. Please continue to report suspected *E. coli O157* infections and send isolates to the State Public Health Laboratory.

Inside this Issue...

Results of the Special Invasive Bacterial Infections Study

Missouri 1992

Marty Huber, R.N.

As reported in the January-February 1992 issue of the *Missouri Epidemiologist*, Missouri is one of six sites participating in active, laboratory-based surveillance for invasive infections caused by *Haemophilus influenzae, Neisseria meningitidis, Listeria monocytogenes*, and Group B streptococcus (GBS). The Special Invasive Bacterial Infections Study, which began in Missouri in January 1992, is being conducted by the Centers for Disease Control and Prevention (CDC). Active surveillance for this study involves review by project staff of microbiology records in each participating hospital/laboratory. All hospitals and laboratories in the state are participating in the study, with the exception of 13 Kansas City hospitals, which, through the Kansas City Health Department, declined to participate.

It was expected that with active surveillance, an increased number of cases of *H. influenzae*, *L. monocytogenes* and meningococcal disease would result, even though these diseases have been reportable by law to the local public health unit or the Bureau of Communicable Disease Control for several years. The number of 1992 cases reported through the usual reporting system and those found by laboratory review are shown in Table 1.

Table 1. Invasive infections by selected pathogens* Missouri 1992.

	Cases Reported to Local or State H.D.	Cases Discovered during Laboratory Review	Total Cases
Haemophilus influenzae			
Meningitis	14	0	14
Other Invasive	30	38 (55.9%)	68
Listeria monocytogenes	15	2 (11.8%)	17
Neisseria meningitidis	36	3 (7.7%)	39

*The number of cases of these diseases in this report will differ from the number of cases reported in the annual report because the study uses the date of culture, whereas other reports use the date the case was reported.

Significant numbers of non-reported cases of *H. influenzae* invasive disease were found on laboratory review. Consequently, comparison of the total number of 1992 cases with the numbers of reported cases from prior years will likely not be valid. However, one can state with reasonable confidence that the advent of Hib vaccine use in 1985, combined with the 1991 recommendation that Hib immunization begin at two months of age, has reduced invasive *H. flu* disease in the state as evidenced by the decrease in *H. flu* meningitis from a high of 172 cases in 1986 to only 14 cases in 1992.

Group B streptococcus has long been known to cause serious invasive disease, and the occurrence of sporadic cases as well as outbreaks of the disease, has often led to intense scrutiny for additional cases for a short period of time. However, when no more cases occurred, interest in GBS was often lost as attention turned to other, more common infections. Reporting of GBS disease has not been required in Missouri or most other states.

Table 2. Adult invasive group B streptococcal disease infections and deaths by race and age group, Missouri 1992

	White		Black		Asian/Pacific Islander		Unknown		Totals	
Age Group	**Cases**	**Deaths**	**Cases**	**Deaths**	**Cases**	**Deaths**	**Cases**	**Deaths**	**Cases**	**Deaths**
15–19 years	7	-	3	-	-	-	-	-	10	-
20–29 years	42	2	25	-	2	-	-	-	69	2
30–39 years	28	-	8	1	-	-	1	-	37	1
40–49 years	21	-	1	-	-	-	-	-	22	-
50–59 years	10	1	5	-	-	-	-	-	15	1
60–69 years	29	3	5	1	-	-	-	-	34	4
70–79 years	34	4	6	-	-	-	-	-	40	4
80–89 years	39	8	3	2	-	-	-	-	42	10
90–99 years	10	4	2	-	-	-	-	-	12	4
Totals	**220**	**22**	**58**	**4**	**2**	**-**	**1**	**-**	**281**	**26**
Percent of total adult cases/deaths	*78.3*	*84.6*	*20.6*	*15.4*	*0.7*		*0.4*		*100*	*100*

Through the Special Invasive Bacterial Infections Study, CDC is seeking to determine the true incidence and nature of GBS invasive disease in a population of approximately 20 million persons. The study began in 1990 in four areas (the state of Oklahoma, metropolitan areas of Atlanta and San Francisco and four counties in Tennessee) having a total population of 10.1 million. (The results of the 1990 surveillance were reported in the *Morbidity and Mortality Weekly Report*, Vol. 41/No. SS-6). Missouri and Maryland were added to the study in late 1991.

During 1992, 412 cases of invasive GBS disease were identified in Missouri. Despite biweekly telephone calls by project staff to all participating microbiology laboratories to collect reports of new cases, 144 (35.0%) were found only through subsequent laboratory record reviews.

GBS disease in Missouri led to 33 deaths, for a case fatality rate of 8.0 percent. Of the 26 adults (≥15 years) who died, only eight were younger than 70 years. Two were in their 20s, one was 35-years-old, one was 54, one 64, two were 67 and one was 68. White persons accounted for 84.6 percent of all adult GBS deaths; blacks accounted for 15.4 percent. See Table 2.

There were 123 cases in infants (<90 days of age) comprising 29.9 percent of the total number of cases of GBS disease. One hundred (81.3%) cases were early onset (<7 days of age). Seven of these infants died or were stillborn, for a case fatality rate of 7.0 percent for early onset disease. All deaths were within two days of birth. Four deaths (57.1%) were in white infants, and three (42.9%) were in black infants, while 86 (69.9%) cases were white and 37 (30.1%) were black. (No other racial groups were represented in infants <90 days of age.) See Table 3.

Gestational age (<31 days of age) is known for 101 cases. Thirty-six (35.6%) were full term (40 weeks) and 10 (9.9%) were 41–42 weeks. Twenty-two (21.8%) were 38-39 weeks gestation, with another 11 (10.9%) being 36-37 weeks gestation. A total of 22 (21.8%) were significantly premature (more than four weeks). Of these, six (27.3%) were less than 30 weeks gestation and four (66.7%) of these babies died or were stillborn.

The complete Missouri 1992 study report will be sent to all Missouri hospitals/laboratories.

Single copies of the report are available upon request from the Bureau of Communicable Disease Control at (314) 751-6113.

Table 3 Infant invasive group B streptococcal disease infections and deaths by race and age at onset, Missouri 1992.

	White		Black		Totals	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Early onset (<7 days)	73	4	27	3	100	7
Late onset (8–90 days)	13	-	10	-	23	-
Totals	**86**	**4**	**37**	**3**	**123**	**7**
Percent of total infant cases/deaths	*69.9*	*57.1*	*30.1*	*42.9*	*100*	*100*

State Public Health Laboratory Report

Newborn Screening — Hypothyroidism, Phenylketonuria, Galactosemia and Hemoglobinopathies

James Baumgartner, B.S., M.B.A., Chief, Metabolic Disease Unit

	Sept. 93	Oct. 93	Total YTD
Specimens Tested	10,357	9,675	98,505
Initial (percent)	64.8%	64.6%	65,690
Repeat (percent)	35.2%	35.4%	32,915
Specimens: Unsatisfactory	139	110	1082
HT Borderline	726	729	7155
HT Presumptive	29	30	249
PKU Borderline	28	10	184
PKU Presumptive Positive	2	2	12
GAL Borderline	28	27	324
GAL Presumptive Positive	3	3	37
FAS (Sickle cell trait)	91	69	930
FAC (Hb C trait)	32	23	280
FAX (Hb variant)	20	14	141
FS (Sickle cell disease)	4	1	24
FSC (Sickle C disease)	2	4	22
FC (Hb C disease)	0	0	0

HT=Hypothyroidism, PKU=Phenylketonuria, GAL=Galactosemia, Hb=Hemoglobin, YTD = Year to Date

Statement on Tuberculosis Screening
January 1994

The following statement was issued by the Missouri Advisory Committee for the Elimination of Tuberculosis (MACET).

In this time of increasing national tuberculosis morbidity and with the looming threat of multi-drug resistant TB facing the nation and Missouri, it is essential that all health care professionals screening for tuberculosis use the most effective tool available.

The Missouri Advisory Committee for the Elimination of Tuberculosis and the Missouri Department of Health strongly advise the use of the PPD Mantoux method as the test of choice when screening individuals for tuberculosis.

The Mantoux PPD test is an intracutaneous test to detect delayed hypersensitivity in persons infected with *M. tuberculosis.*

It is administered by the intracutaneous injection 0.1 ml. of PPD containing 5 tuberculin units on the volar or dorsal surface of the forearm. Reading occurs 48 to 72 hours after injection. The reading should be done by a skilled health care worker or medical professional knowledgeable about the antigen and the technique of administering and reading the test. The reading consists of observing the presence or absence of induration by inspection and palpation of the arm. All reactions shall be measured and recorded in millimeters of induration. Erythema should be disregarded.

When utilizing skin tests to detect TB infection, interpretations should be based upon the Mantoux test only.
Since there is no reliable method of distinguishing between a BCG and tuberculin reaction, individuals with a history of BCG should be tested with a Mantoux PPD, and if found to have a significant reaction, should be considered as indicating infection with *M. tuberculosis.*

More detailed information can be obtained by referring to the Department of Health, Bureau of Tuberculosis Control Manual, and the CDC/ATS guidelines on tuberculin skin testing. The latter is available from the American Lung Association of Eastern and Western Missouri at (800) 586-4872.

Thanks to Diane Rackers, Production Manager

H. Denny Donnell Jr., M.D.

Diane Rackers has been promoted to a Health Program Representative I, and began work with the Hazardous Substances Emergency Events Surveillance (HSEES) program for the Bureau of Environmental Epidemiology in November 1993. She had been employed for 24 1/2 years in secretarial positions with this agency and had served as secretary for Dr. Denny Donnell for 22 of those years. She capably served as production manager for the *Missouri Epidemiologist* since January 1991. Using the Pagemaker program to compose 14 issues and coordinate the work of moving articles from the planning stage through the editorial process to final format and distribution of the published issue. Our sincere thanks and congratu-lations go out to her and we wish her great success with the new endeavor.

Welcome to Jennifer Atkins, Production Manager

H. Denny Donnell, Jr., M.D.

Jennifer Atkins has taken over as production manager with this issue of the *Missouri Epidemiologist.* She is the newly appointed Clerk IV for the Office of Epidemiology as of December 1993. She comes to this position from the health education section of the Bureau of HIV/AIDS Prevention where she assisted in creating various educational prevention materials.

Farewell to EIS Officer, Carol Friedman

H. Denny Donnell Jr., M.D.

Dr. Carol Friedman, who worked with the Missouri Department of Health as a CDC sponsored Epidemiologic Intelligence Officer, completed her two-year assignment at the end of June 1993 and is now working with the CDC in her home state of Texas. In her new post, Dr. Friedman will monitor and promote the progress toward the objectives established for the year 2000. Before leaving Missouri she worked with the St. Louis City Department of Health for a five-month interval developing and supporting the work of the St. Louis Task Force on Syphilis. Her work in Missouri was greatly appreciated and was reflected in the articles she wrote for the *Missouri Epidemiologist.* She was involved in several investigations of outbreaks, provided consultations on a wide range of topics, helped with surveillance issues and helped to edit the Missouri Epidemiologist.

Compliance with Universal Precautions: What Health Care Professionals Need to Know

Caryl Collier, R.N., M.P.H., C.I.C.

As of August 3, 1993, the Centers for Disease Control and Prevention (CDC) was aware of 37 health care workers (HCWs) in the United States who seroconverted from negative to positive for the human immunodeficiency virus (HIV) following occupational exposure to blood and body fluids. Of these 37 HCWs, 32 experienced a percutaneous exposure, 4 had a mucocutaneous exposure and 1 sustained both percutaneous and mucocutaneous exposures. There are 78 other HCWs who are HIV positive or have AIDS who did not report other risk factors for HIV infection but reported occupational exposure to blood, body fluids, or HIV-infected laboratory materials; however, there is no documentation of the pre-exposure HIV status and subsequent seroconversion for these 78 persons.

CDC estimates that approximately 8,000 HCWs become infected with hepatitis B yearly and that approximately 200 HCWs die each year of complications from this infection.

In response to the morbidity and mortality resulting from occupational exposures to blood borne pathogens, the Occupational Safety and Health Administration (OSHA) published its final rule, 29 CFR Part 1910.1030, "Occupational Exposure to Blood Borne Pathogens" in the December 6, 1991, *Federal Register*. OSHA authority extends to all private sector employers with one or more employees as well as federal civilian employers. States administering their own occupational safety and health programs through plans approved under section 18(b) of the Occupational Safety and Health Act of 1970 must adopt standards and enforce requirements that are at least as effective as federal requirements. There are currently 25 states and territories that have their own state plans. Of those, 23 cover the private and public sectors and 2 cover the public sector only. Because Missouri is not one of the states with its own Occupational Safety and Health Office, the public sector is generally exempt from the federal rule except for ambulance workers, who were brought under the OSHA requirement by state rule (see discussion below). Missouri public sector employers include state and local governments which subsidize county health departments, home health organizations, fire safety and law enforcement personnel, schools, hospitals and nursing homes.

Although there is no federal law to mandate public sector employers to comply with the OSHA blood borne pathogens rule, there is state law, 191.650, 191.656, 191.694 RSMo Supplement 1992, requiring all Missouri licensed health care professionals and licensed health care facilities to practice universal precautions as recommended by CDC. This statute also requires that health care professionals and facilities comply with CDC guidelines on disinfection and sterilization of reusable invasive devices. Health care facilities will need to show proof that instruction on infection control procedures has been provided to all patient care personnel. In addition, all health care professionals who perform invasive procedures must receive training, approved by the Missouri Department of Health, on infection control procedures relevant to HIV and related diseases, including universal precautions and prevention of percutaneous injuries.

As of April 26, 1990, all ambulance services were required to follow CDCs "Guidelines for Prevention of Transmission of Human Immunodeficiency Virus and Hepatitis B Virus to Health-Care and Public Safety Workers," MMWR, June 23, 1989, Vol. 38 No. S-6. Effective February 1993, the rule, 19 CSR 30.045, "Communicable Disease Policy" for ambulance services was upgraded to require compliance with the OSHA standard for blood borne pathogens.

19 CSR 30-40.047 (to be published as a final rule next year) will establish mandatory procedures to facilitate fact finding and follow-up recommendations subsequent to communicable disease exposures (possible or definite) incurred by emergency response personnel. The rule will stipulate the appropriate reporting mechanism for emergency response personnel who acknowledge a blood and body fluid exposure. The rule will also stipulate the responsibility of the medical facility and designated officers in notifying emergency response personnel of their possible exposure to certain diagnosed communicable diseases. A companion rule 19 CSR 30-40.048 will mandate relevant training on universal precautions and other related activities. Both rules are authorized by a new section, 192.806-1 RSMo (1993 supplement).

The Department of Health recommends that all public entities establish policies and procedures that meet the requirements of the federal OSHA blood borne pathogens rule. In so doing, an exposure control plan should be written which outlines how the employer intends to eliminate or minimize employee exposures to blood borne pathogens. A generic sample exposure control plan can be obtained from OSHA or the Bureau of Communicable Disease Control (314) 751-6115.

Booklets and fact sheets on OSHA standard 29 CFR Part 1910.1030 can be obtained from OSHA Publications, 200 Constitution Avenue, N.W., Room N3101, Washington, DC 20210 or by calling (202)-219-8148. A videotape can be obtained from OSHA for a fee by calling the National Audio-Visual Center (301) 763-1896.

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Latex Allergies Problematic for Some Patients and Health Care Workers

Caryl Collier, R.N., M.P.H., C.I.C.

Severe latex hypersensitivity (allergy) in several health care workers has been reported to the Missouri Department of Health's, Bureau of Communicable Disease Control. These reports, along with inquiries about alternatives to latex, initiated a literature search and contact with resource persons from the Centers for Disease Control and Prevention (CDC), the United States Food and Drug Administration (FDA), and the American College of Allergy and Immunology (ACAI).

In order to prevent transmission of blood borne pathogens, CDC recommended, and the Occupational Safety and Health Administration mandated, Universal Precautions; consequently, during the past six years, health care workers have been wearing more gloves than ever before. Many of these gloves are made of latex, which provides excellent tactile contact in surgical or examination procedures. A spokesperson from Market Intelligence Research Corporation in Mountain View, California, quotes a 1993 report, "Disposable Medical Products Market Study" (#897-54), by saying that total U.S. medical glove (latex and vinyl) expenditure was $426 million in 1992, up from $385 million in 1989. Projected expenditure in 1999 for latex and vinyl gloves is $782 million. Coincident with an increase in exposure to latex comes the potential for an increase in reports of latex hyper sensitivity.

Allergies to latex have been recognized in patients, and rubber industry workers in many reports from investigators since 1979. As of April 1992, over 600 serious reactions to latex have been reported to the FDA, including at least 16 anaphylactic fatalities. However, investigators are not sure what an increase in reports really mean. Does it mean increased exposure and sensitization to latex? Better diagnosis of latex allergies? Increased protein content of natural rubber because of production methods, i.e., washing of raw latex? It is known that glove antigen content varies from brand to brand and lot to lot. The antigens can be those in the rubber itself as well as those added during production of the commercial product, including powders in gloves. Rubber antigens can be attached to glove powder or cornstarch particles in the gloves.

Researchers estimate that latex allergy exists in less than 1 percent of the general population and 6 percent to 14 percent of health care workers. Unpublished studies indicate that subsegments of health care workers are being identified with much higher latex allergy rates. High risk patient populations are those requiring multiple exposures to latex during medical/surgical procedures. Case reports and surveys reveal a high rate of sensitization in persons with spina bifida/myelomeningocele (FDA reports 18-40%) and persons with urogenital abnormalities, requiring recurrent instrumentation of the genitourinary tract. Also included in high risk patient populations are those receiving barium enema procedures with a latex balloon tip. (The FDA says these balloon tips are no longer being manufactured with latex, but some with latex still remain on the market.) Other patients having allergic reactions to latex include those receiving oral, vaginal and rectal examinations with latex gloves, those with rubber orthodontic appliances, rubber dental dams, those inhaling aerosolized latex and those exposed inadvertently to latex from rubber stoppers and injection ports during intravenous administration. According to the ACAI, the majority of patients who develop latex hypersensitivity have a history of some prior allergic condition, e.g., hay fever. Thus, exposure to latex antigens occurs by direct contact through cutaneous, mucosal, parenteral and inhaled routes.

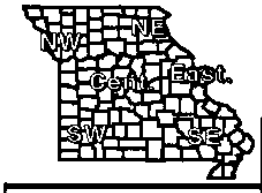
The symptoms of hypersensitivity to latex and/or natural and cured rubber vary from mild eczema or skin rash to hives (local and systemic), rhinitis, conjunctivitis, bronchospasm or asthma and anaphylaxis. Some of these reactions are similar to drug or stinging insect venom responses in which the throat muscles contract and cardiac shock ensues quickly. In some persons, mild symptoms occur upon initial exposure to latex, but after repeated exposures, severe symptoms develop which can lead to death if not quickly recognized and treated.

Preliminary recommendations of the ACAI were published in March 1992. They contain two essential components:

1. **Latex allergy identification:**
   1) a careful history (questions about itching, rash or wheezing after wearing latex gloves, after having a medical procedure performed or inflating a toy balloon) and 2) conduct skin prick tests for latex in experimental settings with protocols to manage anaphylactic reactions. Serum tests for latex-specific IgE, currently performed on a research basis at several laboratories, may confirm a suspected diagnosis in many cases but presently lack sensitivity to identify all patients with true latex allergy.

2. **Latex avoidance procedures:**
   1) "latex-sensitive" wristbands and labeling of patient charts; 2) a list of all products and medical procedures utilizing latex; 3) a list of safe items for latex-sensitive persons; 4) latex-free surgical suites for elective surgery on patients having had latex-caused anaphylaxis and/or myelomeningocele and 5) provision of synthetic latex or non-latex products for health care workers showing signs of latex contact dermatitis or latex hypersensitivity.

(continued on page 9)



Missouri Department of Health
Disease Prevention - Communicable Disease Control
BIMONTHLY MORBIDITY REPORT

Reporting Period *
September/October, 1993

	Districts							KANSAS CITY	ST. LOUIS CITY	ST. LOUIS CO.	SPGFLD GREENE CO.	2 MONTH STATE TOTALS		CUMULATIVE		
	** NW	NE	CD	SE	** SW	** ED	*** OTHER							FOR	FOR	5 YR MEDIAN
Vaccine Preventable Dis.																
Chickenpox	135	35	39	122	68	38		0	0	0	0	437	562	7900	8158	7128
Diphtheria	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Hib Meningitis	0	1	1	2	0	0		0	0	0	0	4	4	9	14	73
Hib Other Invasive	2	1	4	2	0	2		1	3	2	0	17	11	82	30	**
Influenza	1	0	0	0	0	0		0	0	0	0	1	0	250	62	142
Measles	0	0	0	0	0	0		0	0	0	0	0	0	1	0	2
Mumps	3	0	0	0	0	1		0	0	3	0	7	6	32	35	35
Pertussis	11	0	2	2	5	1		0	12	11	0	44	33	123	97	91
Polio	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Rubella	0	0	0	0	0	0		0	0	0	0	0	0	1	1	1
Tetanus	0	0	0	0	0	0		0	0	0	0	0	1	0	1	1
Viral Hepatitis																
A	10	3	12	3	4	7		8	100	58	25	230	364	1293	1028	615
B	5	2	6	3	5	7		9	26	18	5	86	83	402	427	498
Non A - Non B	0	0	0	1	0	0		6	0	0	0	7	5	42	23	43
Unspecified	0	0	0	0	2	0		0	0	0	0	2	0	8	9	14
Meningitis																
Aseptic	21	4	21	7	11	5		6	1	11	7	94	91	233	233	203
Meningococcal	2	0	0	1	1	1		1	2	2	0	10	5	49	20	28
Other	1	1	0	0	0	0		0	3	3	0	8	6	57	37	49
Enteric Infections																
Campylobacter	13	4	8	15	12	9		10	8	23	12	114	129	532	535	470
Salmonella	19	3	13	26	11	13		11	11	17	1	125	86	412	362	590
Shigella	5	0	6	6	1	23		15	8	20	1	85	112	570	652	369
Typhoid Fever	0	0	0	0	0	0		0	0	0	0	0	1	2	2	2
Parasitic Infections																
Amebiasis	0	1	1	0	0	1		7	2	3	0	15	4	44	23	21
Giardiasis	21	4	17	11	13	26		28	8	43	19	190	199	590	625	664
Sexually Transmitted Dis.																
AIDS	8	1	10	2	6	4	1	28	27	27	1	115	148	1528	567	472
Gonorrhea	72	28	94	80	57	19		550	1499	581	0	2980	2708	11161	12433	14869
Genital Herpes	36	17	51	23	66	39		166	143	147	0	688	574	3134	2954	2734
Nongonoc. urethritis	23	12	43	21	5	0		254	589	168	14	1129	1006	5381	5876	6183
Prim. & Sec. syphilis	4	3	0	10	3	5		29	205	58	1	318	234	1243	980	216
Tuberculosis																
Extrapulmonary	0	0	1	1	2	0	0	0	3	2	1	10	4	36	32	33
Pulmonary	2	0	4	6	8	2	1	2	6	4	0	35	30	180	159	169
Zoonotic																
Animal Bites	122	22	69	102	39	70		0	0	503	4	931	1181	5344	5779	3875
Psittacosis	0	0	0	0	0	0		0	0	0	0	0	0	1	1	0
Rabies (Animal)	1	0	0	2	1	0		0	0	4	1	9	16	28	34	31
Rocky Mtn. Sp. Fever	1	0	1	2	1	0		0	0	0	0	5	5	20	24	33
Tularemia	0	1	2	1	0	0		0	0	0	0	4	4	16	34	35

Low Frequency Diseases

Anthrax
Botulism
Brucellosis
Chancroid
Cholera
Cryptosporidiosis
Encephalitis (infectious) - 5
Encephalitis (viral/arbo-viral)
Granuloma Inguinale
Kawasaki Disease
Legionellosis
Leptospirosis
Lymphogranuloma Venereum - 1
Malaria
Plague
Rabies (human)
Reye's Syndrome
Rheumatic fever, acute
Toxic Shock Syndrome
Trichinosis

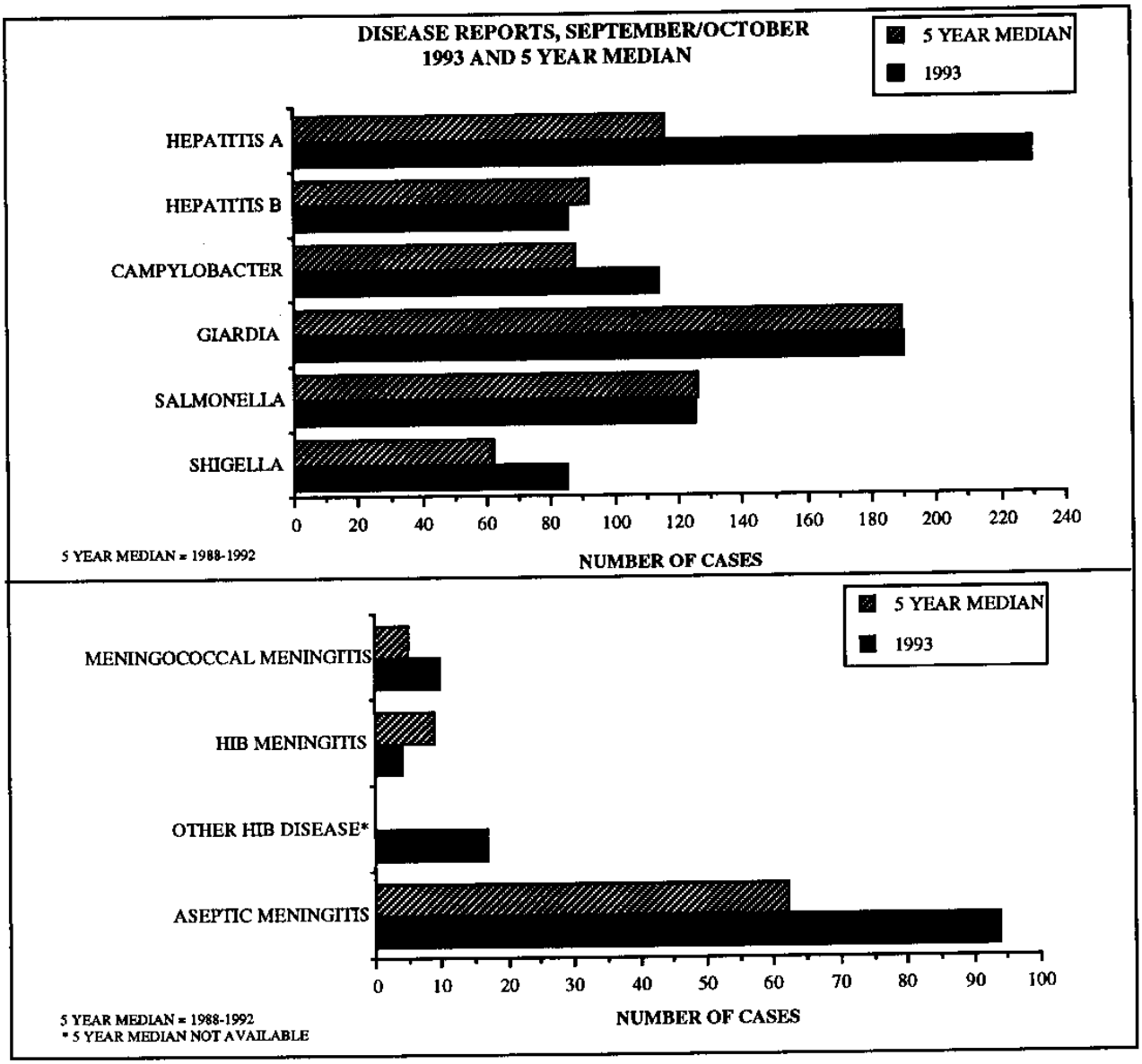
Outbreaks

Foodborne - 1
Waterborne
Nosocomial
Pediculosis
Scabies - 5
Other
Citrobacter bacteremia - 1
Hepatitis A - 2
Shigella - 1

*Reporting Period Beginning August 29, Ending October 30, 1993.
**Totals do not include KC, SLC, SLCo, or Springfield
***State and Federal Institutions
** Data not available

Due to data editing, totals may change.

TEAR OUT FOR FUTURE REFERENCE



VIRAL HEPATITIS

Hepatitis A decreased 36.8%, from 364 cases in September/October 1992 to 230 during the same period in 1993. This is an increase of 98.3% from the five year median of 116 cases. Hepatitis B, at 86 cases, is up 3.6% from 83 cases in 1992. This is an increase of 6.5% from the five year median of 92 cases.

ENTERICS

Campylobacter decreased 11.6%, from 129 cases in 1992 to 114 in 1993. This is an increase of 29.5% from the five year median of 88 cases. Salmonella, at 125 cases, is up 45.3% from 86 cases in 1992 and down 0.8% from the five year median of 126 cases. Shigellosis decreased 24.1% from 112 cases in 1992 to 85 in 1993. It increased 37.1% from the five year median of 62 cases.

PARASITES

Giardiasis, at 190 cases, is down 4.5% from 199 cases in 1992. It is 0.5% above the five year median of 189 cases.

MENINGITIS

Aseptic meningitis increased 3.3%, from 91 cases in 1992 to 94 in 1993. This is an increase of 51.6% from the five year median of 62 cases. Meningococcal meningitis increased 100%, from 5 cases in 1992 (which is also the five year median) to 10 cases in 1993.

HIB DISEASE

There were 4 cases of Hib meningitis reported for the1993 period, which is the same as 1992. This is down 55.5% from the five year median of 9 cases. The Bacterial Infections Surveillance Project continues to affect reporting of other invasive Hib disease; it increased 54.5%, from 11 cases in 1992 to 17 in 1993. There is no five year median available for other invasive Hib disease

Latex Allergies

(continued from page 6)

Active research efforts to understand the causes and treatment of latex allergy are continuing and new findings are anticipated by ACAI. One researcher of latex allergy is Dr. Jay Portnoy, Chief of Allergy at Children's Mercy Hospital in Kansas City. Dr. Portnoy is testing (when indicated) for latex allergy in employees and in children under 18 years of age who are inpatients or outpatients. Dr. Portnoy echoes the FDA Medical Alert: "Allergic Reactions to Latex-Containing Medical Devices," March 29, 1991, in saying that hypoallergenic gloves might be satisfactory for latex allergic health care workers, but latex-free gloves are necessary for contact with latex-allergic patients. The Task Force for Latex Allergy at Children's Mercy Hospital has developed a comprehensive set of recommendations, which were revised July 16, 1993, for identifying and managing persons with latex-allergies. For more information, persons may call (816) 234-3097.

Cotton liners for gloves and non-latex surgical/examination gloves are available from several manufacturers, as are non-rubber bladder catheters, enema tubes, endotracheal tubes, paper tape, clear plastic face masks, and non-latex tourniquets. A health care worker could wear a non-latex glove over the latex glove if the patient is sensitive. If both the health care worker and patient are sensitive, a latex middle glove could be used. FDA is recommending double gloving when vinyl gloves are necessary because of greater porosity occurring in the vinyl manufacturing process. However, CDC does not recommend latex rather than vinyl gloves when quality control standards are equal.

Some authors recommend that a regimen of medication with corticosteroids, H_1 antagonists, ephedrine, and sometimes H_2 antagonists precede radiography with contrast media. This same regimen has been recommended by other authors to be used preceding major surgical and dental procedures in latex-allergic patients; however, Dr. E. Slater, author of, "Allergic reactions to natural rubber," Annals of Allergy, Vol. 68, March 1992, warns that anaphylaxis has occurred in rubber-sensitive individuals in spite of this regimen; consequently, the regimen cannot be an alternative to antigen avoidance.

Plant physiologists, such as Katrina Cornish of USDA's Agricultural Research Service, are working on allergen-free rubber extracted from the stems and bark of the guayule bush which grows wild in Arizona and Mexico. The rubber from this bush appears to be free of the allergy-causing proteins found in latex made from the tropical rubber tree. If research shows this to be the case, then products such as medical gloves, toys and clothing could be manufactured free of latex. However,even if this is possible, there is always the potential, according to Sharon Dillard of the FDA, that allergy-prone individuals will develop sensitivities to the proteins in a new naturally-derived product.

Soon to be published in the *Federal Register* for public comment is the FDA decision to require labels on products containing latex, thus alerting latex-allergic persons to avoid the products. All reactions to latex should be reported to the FDA at (800) 638-6725.

The Health Industry Manufacturers Association (HIMA) and the CDC are collaborating on a study of reactions to latex-containing products. The purpose of this study is to determine the prevalence and type of natural rubber latex reactions among workers at a wide variety of health care facilities and natural rubber latex manufacturing plants nationwide.

HIMA and CDC are now actively soliciting health care facilities and organizations for participation in this study.

For more information on the project and participant responsibilities, contact:
Siiri N. Bennett, M.D.
Hospital Infections Program
MS A-07
Centers for Disease Control
and Prevention
1600 Clifton Road NE
Atlanta, GA 30333
Phone: (404) 639-1550
Fax: (404) 639-3770

Barry Page, Director
Environmental Technology Programs
Health Industry
Manufacturers Association
1200 G. Street NW, Suite 400
Washington, DC 20005

For more information, contact Caryl Collier at (314) 751-6115.

Universal Precautions

(continued from page 5)

Consultation on compliance with the OSHA standard can be obtained from the following sources: Missouri Onsite Safety and Health Consultation Services, Division of Labor Standards at (314) 751-3403; Missouri Department of Health's, Bureau of Dental Health at (314) 751-6247; Missouri Department of Health's, Bureau of Communicable Disease Control, 314-751-6115; Kansas safety and health consultation: 913-296-4386. Consultation can also be obtained from the OSHA area offices - Kansas City: (800) 892-2674 or (816) 483-9531; St. Louis area office: (800) 392-7743 or (314) 425-4249 and from the OSHA regional office: (816) 426-5861.

Notice to Readers:

The previous issue of the *Missouri Epidemiologist* was July-October. The *Missouri Epidemiologis*t was not issued for November-December.

Update: Hantavirus Pulmonary Syndrome - United States, 1993

Reprinted in part from MMWR, Vol. 42/No. 42, October 29, 1993, and MMWR Vol. 43/No. 3, January 28, 1994

In June 1993, a newly recognized hantavirus was identified as the etiologic agent of an outbreak of severe respiratory illness (hantavirus pulmonary syndrome [HPS]) in the southwestern United States. Since this problem was recognized, sporadic cases have been identified from a wide geographic area in the western United States.

Through December 31, 1993, 53 persons with illnesses meeting the surveillance case definition of HPS have been reported to the Centers for Disease Control and Prevention. Patients' ages have ranged from 12 years to 69 years (median age: 31 years), and 32 (60%) were aged 20-39 years; 30 (57%) were male. Twenty-six (49%) were American Indians; 22 (42%), non-Hispanic whites; four (8%), Hispanic; and one (2%), non-Hispanic black. Thirty-two (60%) patients died; persons with fatal cases and persons with nonfatal cases were similar in age, sex, and race.

Cases have occurred in residents of 14 states including New Mexico (18), Arizona (11), Colorado (5), California (3), Nevada (3), Idaho (3), Montana (2), South Dakota (2) and Oregon, North Dakota, Minnesota, Texas, Kansas, and Louisiana with one each. Of the 34 (64%) persons who were residents of Arizona, Colorado, or New Mexico, illness occurred in 25 (74%) during April - July 1993 and in one before 1993. In comparison, of 19 cases reported from other states, five (26%) had onset of illness during April - July 1993, and seven (37%) had onset before 1993. All patients either lived in rural areas or had visited rural areas during the 6 weeks before onset of illness.

The etiology of HPS was initially identified by serology, polymerase chain reaction (PCR), and immunohistochemistry. Additional cloning and sequencing of virus ribonucleic acid (RNA) from human autopsy tissues indicated that all three of the RNA segments of this new virus were unlike those of any known hantavirus; the new hantavirus is most closely related to the Prospect Hill strain of hantavirus.

In November 1993, the etiologic hantavirus associated with HPS was isolated from tissues of a deer mouse (*Peromyscus maniculatus*) trapped in New Mexico in June 1993 near the residence of a person with confirmed HPS. Lung material from this animal was twice passed in uninfected laboratory deer mice and then adapted to Vero E6 cell cultures. The genetic sequence of the 139-nucleotide PCR product from the isolated virus was identical to PCR products amplified from this rodent in June 1993 and from lung tissue of the associated patient. At the same time, the U.S. Army Medical Research Institute of Infectious Diseases isolated the virus from specimens from a person in New Mexico and from a rodent in California. Muerto Canyon virus has been proposed as the name for this virus, following standard conventions for naming zoonotic viruses after a nearby geographic feature.

The prognosis is poorest in case-patients with shock and with severe lactic acidosis. Anecdotal reports suggest that periods of severe hypoxia or hypotension before stabilization in the intensive-care setting adversely affect survival. Supportive measures are the basis for therapy; severe hypoxia and overhydration should be avoided or prevented. Pressors or cardiotonic drugs should be employed to maintain perfusion without excessive fluid administration. Testing for alternative diagnoses should be done, and appropriate treatment to cover infections mimicking HPS should be administered early. Serologic tests in combination with PCR and IHC should be used in confirming the diagnosis of acute hantavirus infection.

No defined set of symptoms and signs reliably distinguishes HPS at the time of presentation from other forms of non-cardiogenic pulmonary edema or adult respiratory distress syndrome. Onset of illness has been characterized by a prodrome consisting of fever, myalgia, and variable respiratory symptoms (e.g., cough) followed by the abrupt onset of acute respiratory distress. Other symptoms reported during the early phase of illness have included headache and gastrointestinal complaints. Other findings include thrombocytopenia, hemoconcentration, leukocytosis, increased band forms on differential, hypoalbuminemia, and lactic acidosis. In all case-patients reviewed, bilateral pulmonary infiltrates developed within 2 days of hospitalization. The hospital course was characterized by fever, hypoxia, and hypotension; recovery in survivors has been without sequelae.

Previously isolated hantaviruses have demonstrated in vitro sensitivity to the investigational antiviral drug ribavirin. Based on this finding and evidence of activity of intravenous ribavirin therapy against Hantaan virus infection, intravenous ribavirin has been made available as an investigational agent though a CDC-sponsored open label protocol for patients with HPS. Whether treatment with ribavirin has had any beneficial effect on the course of HPS is unknown. Further plans for study of ribavirin are under consideration by a collaborative working group sponsored by the National Institute for Allergy and Infectious Diseases, National Institutes of Health. Physicians who want to enroll patients should contact the CDC Ribavirin Officer of the Day at (404) 639-1510 weekdays or (404) 639-2888 evenings and weekends. Physicians must report patients meeting the screening criteria for HPS and submit appropriate clinical samples to state health departments to confirm the diagnosis.

New clinical syndromes and infections associated with previously unknown pathogens often are recognized only after clinicians and public health officials

become aware of clusters of cases. In May and June 1993, the recognition and reporting of 24 cases of severe respiratory illness among residents of the southwestern United States led to a multiagency response that included state and local health departments, universities, the Indian Health Service, the Navajo Nation Division of Health, and CDC. This response, in turn, resulted in the identification of HPS.

Disease associated with hantaviruses occurs primarily in otherwise healthy adults; however, HPS affects both sexes while infection by other hantaviruses affects predominantly males. The case-fatality rate for persons infected with Muerto Canyon virus has been substantially (more than 10 times) higher than that for persons infected with other hantaviruses. Factors accounting for the seasonal pattern of HPS have not been fully defined.

Although all confirmed cases of PHS in 1993 occurred in persons who resided west of the Mississippi River, the primary reservoir of the virus, the deer mouse, inhabits all areas of the United States except the southeast and Atlantic seaboard. Since January 1, 1994, one case of HPS has been confirmed in a resident of Indiana, and a possible case is under investigation in Florida. Regional variations in the occurrence of this problem and observed differences in the racial/ethnic and age distribution may reflect differences in 1) activities associated with exposure or transmission, 2) local surveillance and retrospective case finding, or 3) the prevalence of the virus in the rodent host. For example, persons participating in agricultural activities near habitats of infected rodents are likely to be at greater risk for infection.

Screening Criteria for Hantavirus Pulmonary Syndrome in Persons with Unexplained Respiratory Illness Potential case-patients must have on eo fthe following:
Potential case-patients must have one of the following:

- a febrile illness (temperature > 1010 F [>38.30 C]) occurring in a previously healthy person characterized by unexplained adult respiratory distress syndrome, OR bilateral interstitial pulmonary infiltrates developing within 1 week of hospitalization with respiratory compromise requiring supplemental oxygen,

- an unexplained respiratory illness resulting in death in conjunction with an autopsy examination demonstrating noncardiogenic pulmonary edema without an identifiable specific cause of death.

Potential case-patients are to be excluded if they have any of the following:

- a predisposing underlying medical condition (e.g., severe underlying pulmonary disease, solid tumors or hematologic malignancies, congenital or acquired immunodeficiency disorders, or medical conditions [e.g., rheumatoid arthritis or organ transplant recipients] requiring immunosuppressive drug therapy [e.g., steroids or cytotoxic chemotherapy]).
- an acute illness that provides a likely explanation for the respiratory illness (e.g., recent major trauma, burn, or surgery; recent seizures or history of aspiration; bacterial sepsis; another respiratory disorder such as respiratory syncytial virus in young children; influenza; or legionella pneumonia).

Confirmed case-patients must have the following:

- at least one specimen (i.e., serum and/or tissue) available for laboratory testing for evidence for hantavirus infection.
- in a patient with a compatible clinical illness, either serology (presence of hantavirus-specific immunoglobulin M or rising titers of immunoglobulin G), polymerase chain reaction for hantavirus ribonucleic acid, or immunohistochemistry for hantavirus antigen is positive.

Recognition of the more geographically widespread occurrence of HPS emphasizes the need for physicians and other health-care providers to consider this problem in the differential diagnosis of adult respiratory distress syndrome. Cases meeting the clinical screening criteria should be reported to CDC through the Missouri Department of Health.

New Epidemic Strain of *Vibrio Cholerae*

Mahree Fuller Skala, M.A.

A newly identified strain of *Vibrio cholerae* has been responsible for tens of thousands of cases and hundreds of deaths in India and Bangladesh since October 1992. The Centers for Disease Control and Prevention (CDC) reported the first case imported into the U.S. in July 1993 (1).

The strain responsible for the epidemic does not match any of the previously known 138 types of *V. cholerae*, and has therefore been designated serogroup O139. Previously, it was thought that only *V. cholerae* O1 was capable of causing epidemic disease. The World Health Organization recently expanded the surveillance definition of cholera to include illnesses due to O139.

The symptoms of *V. cholerae* O139 infection are reportedly indistinguishable from those caused by O1 cholera, and are managed with the same oral and intravenous rehydration therapy. The organism has been susceptible to tetracycline, but resistant to trimethoprim-sulfamethoxazole and furazolidone.

The identification of this new pathogen has several important implications for public health. First, the disease has spread rapidly among all age groups in southern Asia, implying that the organism is encountering an "immunologically naive" population (2). This means that further spread in the developing world (and importation to the U.S.) is likely. Prior exposure to *V. cholerae* O1 does not appear to provide protection; neither do the currently available cholera vaccines.

(Continued on page 13)

Investigation of Epidemic Neurodermatitis in Two Elementary Schools

Pat Phillips, D.V.M., M.S.P.H.

In February and March, the Missouri Department of Health had the opportunity to investigate two outbreaks of skin rash among elementary school students in Dixon and Cuba, respectively (See Figures 1 and 2). In both instances, the outbreak was characterized by a pruritic rash that disappeared within 15 minutes of leaving the school building and predominantly affecting female students. Both investigations centered on identifying specific environmental, chemical and biological factors associated with disease incidence.

In Dixon primary environmental factors of interest included CO_2 concentrations, relative humidity and general housekeeping in the school. Carbon dioxide levels were found to be consistently at or above 1000 ppm, which is not toxic but is threshold for indication of inadequate ventilation. Conversely, relative humidity was almost always less than 40 percent, whereas the desirable level is 40 percent to 60 percent. In reviewing the housekeeping functions, a number of shortcomings were noted, e.g. furnace air filters missing or clogged with dust, minor propane leaks at connections with heaters, bird droppings and nesting material in the plenum above a dropped ceiling. Additionally, a large variety of janitorial products were found in the school; when questioned about cleaning practices, it was determined that label directions were not being followed and solutions were being formulated with inordinately high concentrations of active ingredients.

Demographic and specific exposure data were collected for each child in the Dixon school. Analysis of this information showed that 9 to10-year-old-girls had a 50 percent increased risk of having rash. Exposure to new playground equipment, new treated landscaping timber, school-prepared meals, or enrollment in remedial classes were not associated with occurrence of rash.

In Cuba, the same environmental factors (CO_2, relative humidity, and cleaning products) were investigated. Carbon dioxide levels built up rapidly during the day, often exceeding 1000 ppm by mid-morning. Relative humidity was found to be in the acceptable range of 40 percent to 60 percent on all samplings. Cleaning products on site were centrally stored and fewer in number; maintenance personnel observed and followed label directions for each specific product and were careful to use the product only as intended, e.g. floor waxing or stripping, blackboard cleaning, toilet bowl disinfecting.

Again, demographic and exposure information was collected for a group with rash and a randomly selected control group by individual interview of each participant. As before, young girls were found to be at increased risk (69 percent) of rash; additionally, it was determined that if a student's best friend had rash, then that student had an increased risk (77 percent) of having rash. Lastly, exposure to school meals or hand-washing soap, noticing unusual odors, riding a school bus, or having allergies were not found to be related to rash formation.

In these two instances of rash in elementary level students, no indication of biological or environmental cause was identified. No known infectious disease has symptoms that disappear and re-appear within minutes, or afflicts one gender at such a preferential rate. Similarly, there is no known environmental toxicant with these disease characteristics. But there is one condition reported in the medical literature that fits these findings exactly: epidemic neurodermatitis. The mechanism for the development of epidemic neurodermatitis revolves around a visual stimulus resulting in the host mimicking the observed condition. This is analogous to "feeling" skin sensations after seeing fleas on a pet. As the localized rash develops, pruritis and the resultant scratching cause release of more histamine and an increase in size of the area affected. There are no permanent ill-effects, physically or neurologically, after cessation of symptoms.

The frequency of symptoms decreased over a period of a few days as changes were made in ventilation and housekeeping procedures and as the results of investigation showing no environmental cause for alarm were provided to the school personnel.

Recommendations were made to school officials to hopefully prevent future epidemic neurodermatitis incidents. Recommendations revolved around eliminating potential sensory stimuli, and are as follows:

1. Routine, periodic examination of heating and cooling units for mechanical condition, operating efficiency and filter replacement as per manufacturer's guidelines. This will help reduce dust concentrations in the school and prevent the development of small fuel leaks between heating seasons.

2. Increase fresh air intake (in cold weather this may increase the need for additional humidification to maintain a minimum 40 percent relative humidity level).

3. Clean air outlet vents and return air plenum above dropped ceiling.

4. Decrease dust concentrations in the building by more frequent wet mopping and vacuuming. This will also help reduce dust in the classrooms and halls which may be disturbed by normal activities in the building.

5. Continue to use janitorial supplies only as specified in label directions for preparation and application. Following this recommendation will help to reduce the possibility of unwanted

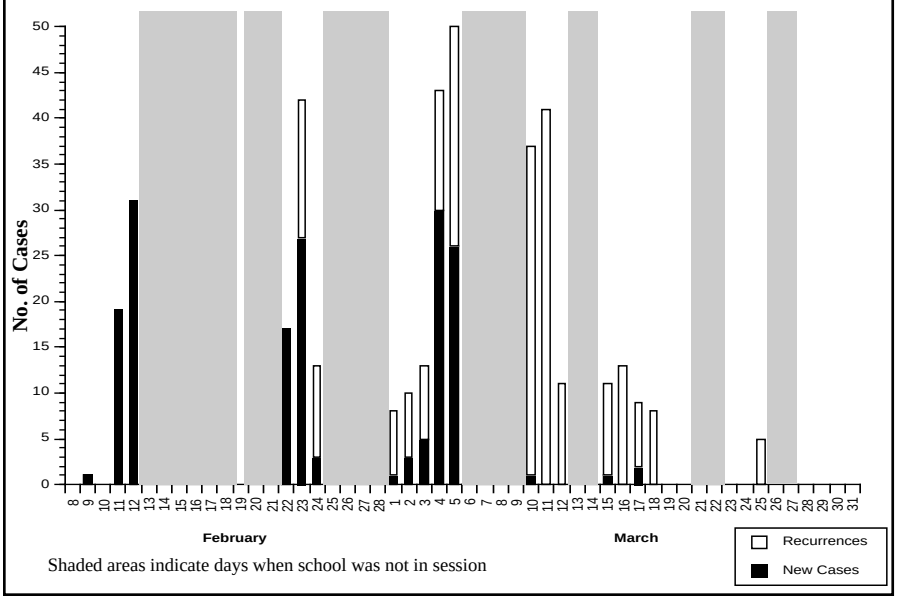
side-effects from chemical components of the supplies in those students and staff who might come in contact with any residue.

6. Promote use of skin moisturizing solutions with vanishing cream base before and during the winter season. This preventive measure will help to reduce dry skin and not leave a visible sign which could suggest a dermatologic condition and reflex rash formation in others.

7. Use non-marking, anti-pruritic compounds, applied in the classroom, in the event of itch or rash symptoms appearing. Relieving the itch sensation should result in less scratching and less rash formation and spread.

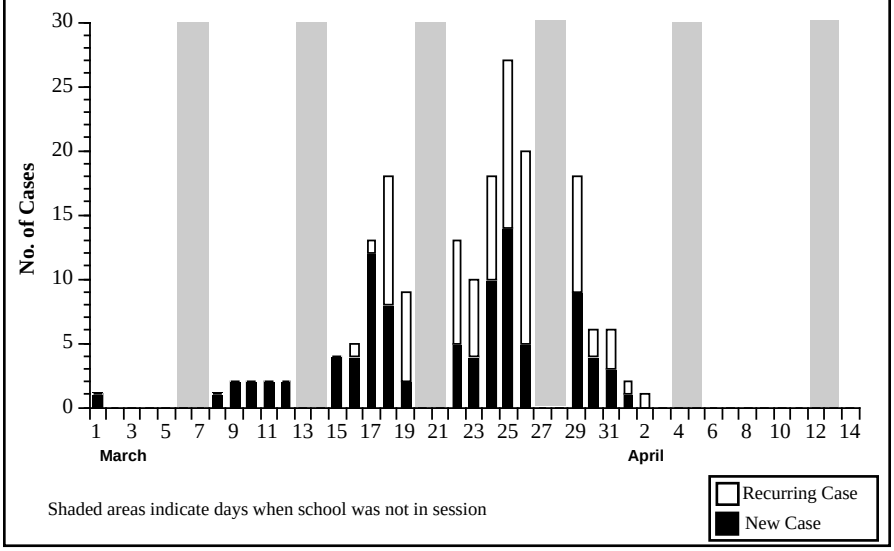
8. School personnel (teachers, administrative and support staff) should be advised to resist disruption of normal routine if rash or itch symptoms reappear. While all medical complaints should be evaluated by the school nurse, unless an emergency condition exist, visits to the nurse's office should be made during non-academic times, e.g. recess, lunch period, before/after school.

Figure 1 Occurrence of Rash Among Dixon Elementary Students, 1993



Shaded areas indicate days when school was not in session

Figure 2 New and Recurring Cases, Cuba Elementary School Rash, 1993



Shaded areas indicate days when school was not in session

New Epidemic

(Continued from page 11)

Second, surveillance for the new organism is difficult because conventional laboratory methods do not distinguish O139 from other unrelated non-O1 *V. cholerae* isolates.

CDC and the Missouri Department of Health recommend the following approach:

1. Report all cases of suspected cholera immediately to your local health department, especially if there is an epidemiologic link to an affected area of the world;

2. Culture specimens from persons with suspected cholera on thiosulfate-citrate-bile salts-sucrose (TCBS) medium for isolation of *V. cholerae*. This testing is also available from the Missouri State Public Health Laboratory (MSPHL); and

3. All clinical isolates of *V. cholerae* should be referred to the MSPHL for further characterization. This is especially true if the illness is typical of severe cholera, is linked to an affected area of the world, or is linked to an outbreak of diarrheal disease.

References

1. CDC. Imported cholera associated with a newly described toxigenic Vibrio cholerae O139 strain—California. MMWR 1993;42:501-503.

2. Anonymous. New cholera subtype breaks rules, poses threat. ASM News 1993;9:430.

Influenza Update

Mahree Fuller Skala, M.A.

A total of 117 laboratory confirmed cases of influenza have been reported as of January 14, 1994. All are type A with 33 subtyped as A/Beijing (H3N2). Table 1 reflects influenza cases by district, county of residence, and type. An age breakdown of cases is shown in Table 2.

As of January 14, 1994, a total of 8 schools had reported outbreaks of influenza-like illness. School closings due to influenza-like illness were reported in 5 schools. The schools are located in Stone, Hickory, Carter, Howell and Texas Counties. Influenza-like illness outbreaks have also been reported in nursing homes in St. Charles, St. Louis, Phelps and Oregon counties; institutions in Callaway and Cole counties; and a community outbreak in Putnam County. No additional outbreaks or school closings have been reported since January 14. We continue to assess the influenza activity in our state as "widespread".

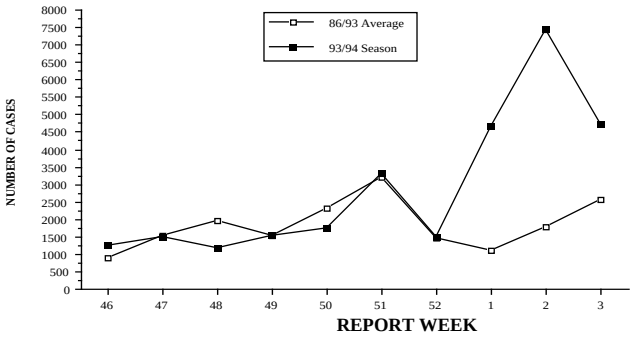
Influenza-like illness, as noted in Figure 1, decreased in week 3. This may be due to numerous school closings due to bad weather (schools serve as surveillance sites). The pneumonia and influenza deaths are fluctuating around the previous 11 year average (Figure 2).

During the 1992-93 flu season, there were 25 reports of school closings due to influenza-like illness. 83 percent of the flu reported during the 1992-93 season was type B which is not usually associated with high levels of mortality. The influenza vaccine for the 1993-94 season includes A/Texas 36/91, A/Beijing 32/92, and B/Panama 45/90.

Table 1 Influenza cases by district, county of residence and type, Missouri 93/94

District	County	Type A (not subtyped)	Type A Beijing (H3N2)	Total
Eastern	Franklin	1	0	1
	Jefferson	4	0	4
	St. Charles	4	3	7
	St. Louis City	16	0	16
	St. Louis County	28	3	31
	Unknown	18	5	23
Central	Boone	4	3	7
	Callaway	0	2	2
	Miller	1	0	1
	Moniteau	0	1	1
	Phelps	0	1	1
	Warren	0	1	1
	Washington	1	0	1
Southeast	Cape Girardeau	1	0	1
	New Madrid	1	0	1
	St. Francois	1	0	1
	Texas	0	1	1
Southwest	Greene	1	0	1
Northeast	Clark	1	0	1
	Knox	0	2	2
	Lewis	0	4	4
	Livingston	0	1	1
	Marion	1	0	1
	Putnam	0	2	2
Northwest	Jackson	0	3	3
	Johnson	1	0	1
	Kansas City	0	1	1
TOTAL		84	33	117

Figure 1 Influenza-like Illness by Week of Report Missouri 83/93 Average 93/94 Season



NOTE: Week 2 and 3 include 5 of 6 districts

Figure 2 Pneumonia and Influenza Dealths Missouri 83/93 Average, 93/94 Season

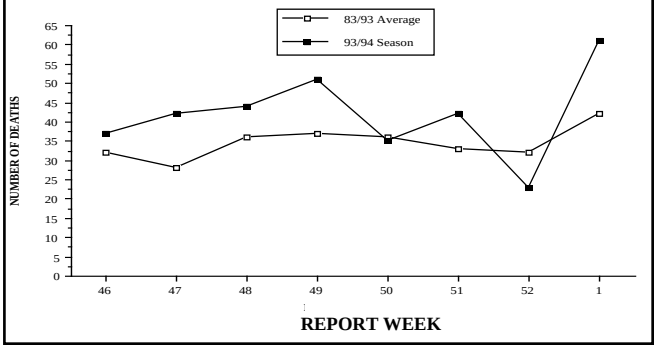


Table 2 Confirmed Influenza Cases

Age group	# of Cases
<1	29
1 - 4	28
5 - 9	9
10 - 14	6
15 - 19	3
20 - 29	8
30 - 39	1
40 - 49	8
50 - 59	3
60 +	18
Unknown	4
TOTAL	117

Hepatitis A Decreased in 1993

Mahree Fuller Skala, MA..

Preliminary data on Hepatitis A incidence in Missouri for 1993 is slightly lower than in 1992, with 1415 cases reported. Figure 1 shows the monthly pattern of reporting for the past three years. Of the 1993 total, 1116 cases (78.9%) were reported from the St. Louis area. The case rate in the Eastern District was 60.2/100,000 for 1993, compared with a rate of 9.2/100,000 in the rest of the state. County-specific rates are shown in Figure 2.

There are indications that the outbreak in the St. Louis area has passed its peak and is gradually declining. Figure 3 shows the number of cases reported in the Eastern District each month since January 1992, compared with the average number each month in 1988-1991. Although hepatitis A activity still slightly exceeds pre-outbreak levels, this trend reflects the coordinated efforts of the local health departments in the area and is very encouraging.

Once hepatitis A increases dramatically in a community, it may take years to return to endemic levels. The Kansas City Health Department recently noted in its "Community and Hospital Letter," September 1993, that although the disease peaked there at 331 cases in 1988, it has not yet returned to the pre-outbreak levels of 1985-86.

Control of hepatitis A depends on prompt diagnosis and reporting of cases. Immune globulin must be administered to contacts within 14 days of exposure to effectively prevent transmission. Please report all suspected cases immediately to your local health department so that contacts may be identified and treated as soon as possible.

Figure 1 **HEPATITIS A REPORTS, BY MONTH MISSOURI 1991-1993**

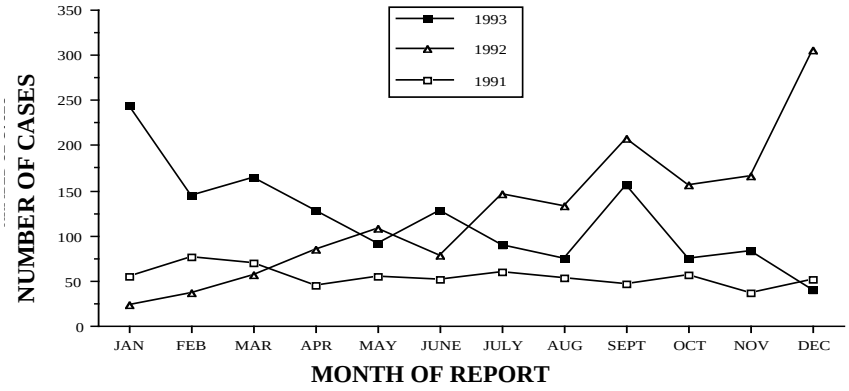


Figure 2 **HEPATITIS A RATES IN MISSOURI COUNTIES 1993**

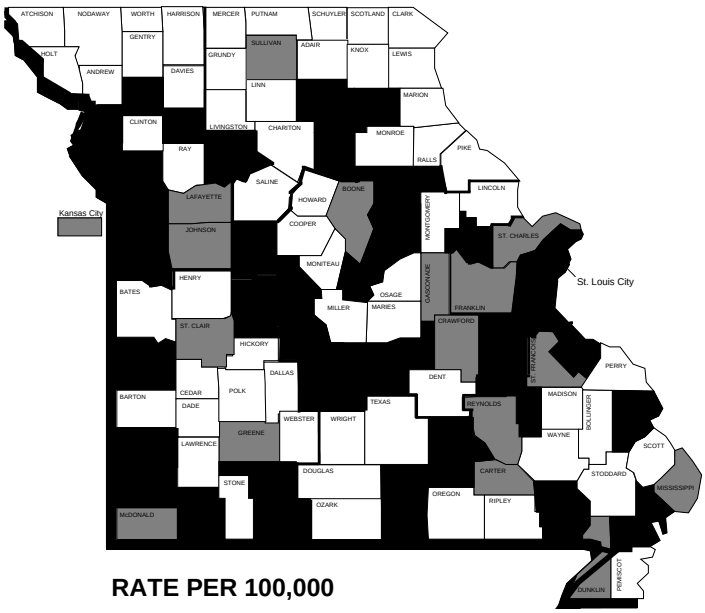
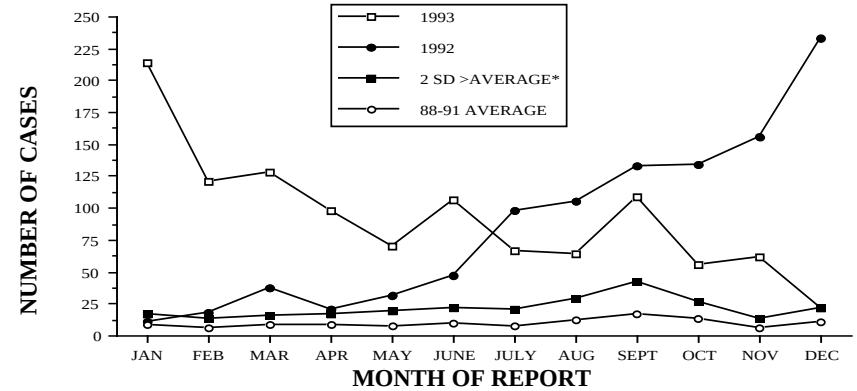


Figure 3 **HEPATITIS A REPORTS, BY MONTH, EASTERN DISTRICT 1988-91 AVERAGE, 1992-1993**



*2 standard deviations >1988-91 Average

Missouri
EPIDEMIOLOGIST

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Managing Editor is H. Denny Donnell, Jr., M.D., M.P.H., State Epidemiologist, assisted by an Editorial Board including Bill Schmidt, M.P.H., Director, and Hilda Chaski, M.P.H., Deputy Director of the Division of Environmental Health and Epidemiology. Jennifer Atkins is Production Manager. Questions or comments should be directed to the Office of Epidemiology at (314) 751-6128 or toll free (800) 392-0272.

Cardiovascular Disease Risk Reduction Conference

May 4-6, 1994
Kansas City, Missouri

"Public Health Partnerships for the Year 2000"

The Missouri, Iowa, Nebraska and Kansas departments of health along with the American Heart Association - Missouri Affiliate and the National Heart, Lung and Blood Institute, are planning the ninth biennial region VII cardiovascular health conference. "Partnerships in Prevention for the Year 2000" will take place at the Plaza Hilton Hotel in Kansas City, Missouri, May 4-6, 1994.

The program is designed for nurses, physicians, dietitians, health educators, administrators, and educators. It will disseminate the latest information on prevention and medical management of cardiovascular disease. It will also be an opportunity for cardiovascular health programs within the region to showcase their initiatives (abstracts are being accepted until March 15, 1994).

For more information on the conference or for abstracts, contact Sue Dabney at:

Missouri Department of Health
201 Business Loop 70 West
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Missouri EPIDEMIOLOGIST

Volume XVII, Number 2 *March-April 1994*

Waterborne *Salmonella* Outbreak in Southeastern Missouri

by Mahree Fuller Skala, M.A., Bureau of Communicable Diseases Control

The investigation of several culture-confirmed cases of *Salmonella* serotype *typhimurium* reported in late November and early December 1993, led to identification of a community-wide outbreak caused by contamination of the public water supply in Gideon, Missouri.

Introduction

On November 29, 1993 a hospital in Butler County reported two inpatients with *Salmonellosis*, both teenagers from Gideon. Active case finding by the southeastern district communicable disease coordinator identified five additional cases within the next two days. Although hospitalized in several different locations, all were associated with Gideon. The State Public Health Laboratory identified several of the isolates as *Salmonella* serotype *typhimurium* with a dulcitol negative biochemical marker, indicating a probable common source outbreak was occurring. An investigation was started immediately.

Background

Gideon is located in New Madrid County, in the "Bootheel" area of southeast Missouri. The population of Gideon is 1,104, with 495 people living in the surrounding township. The topography of the area is very flat. The town has one part-time family practice clinic, one pharmacy, a nursing home, and a school.

The municipal water system was originally constructed in the 1930's. Two deep wells provide the water, which was not chlorinated. The water system was not routinely flushed, but on Nov. 10 it had been flushed systematically by opening each of the fire hydrants in sequence.

Contaminated food is the source of most *Salmonella* infections. It has long been recognized that *Salmonella* can be transmitted by drinking water, but no waterborne outbreaks of the disease had been reported in the U.S. in seven years. The last two such outbreaks were reported in 1986 in Utah and Mississippi; both were attributed to contaminated community water supplies with well water sources [1].

Initial Investigation

Investigators from the Southeastern District Office and the New Madrid County Health Department first focused on the patients' food consumption histories. The reported cases were all children aged 3 to 18 or adults over 60, so special attention was paid to food served at the school and the nursing home. Besides a standard three-day food history, patients were questioned about their food sources (groceries, delis, restaurants) and group activities such as church, organizations, day care, attendance at athletic events, and other local events including a craft fair and a Thanksgiving meal at the nursing home. No commonality could be identified except association with Gideon, which suggested that municipal water might be the source of the outbreak.

On Dec. 15 the Department of Health (DOH) investigators contacted the Department of Natural Resources (DNR) and shared the information they had collected. Routine water testing records were reviewed; samples submitted from the municipal water supply during 1992-93 were all reported safe. DNR representatives went to Gideon on Dec. 16 to take water samples.

At that time they discovered that a valve was open between the municipal water system and a water tower used for fire protection by a local business. The city government had thought that the valve had been closed two years before. Inspection of the tower revealed extremely turbid water, holes in the tower walls, and sheets of rust floating on the water surface.

A boil water advisory was issued by the city at DNR's request on Dec. 18 because coliform bacteria were detected in several water samples taken at the school and from local homes. Fecal coliforms were detected in some of the samples.

(continued on page 2)

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On Dec. 22 the city's two water towers and the fire protection tower were shock treated" with ten gallons of chlorine each, and emergency chlorination equipment was installed at one of the city wells. DNR and DOH representatives collected more water samples from city hydrants on Dec. 23 and did not find chlorine in any of the samples. One of the fire hydrant samples was positive for dulcitol negative *Salmonella* serotype *typhimurium*.

A total of 27 patients with laboratory confirmed salmonellosis was identified. All 21 isolates tested by the SPHL were confirmed as the outbreak serotype. Thirteen of the 27 were hospitalized, and four died. All of the culture confirmed patients were exposed to Gideon municipal water. Ten lived outside Gideon, but eight of them attended school in town, and the other two worked or attended day care there. The first confirmed case had onset of illness on Nov. 17 (see Figure 1). One additional case occurred in January, in a person who had just moved into a previously vacant house in town.

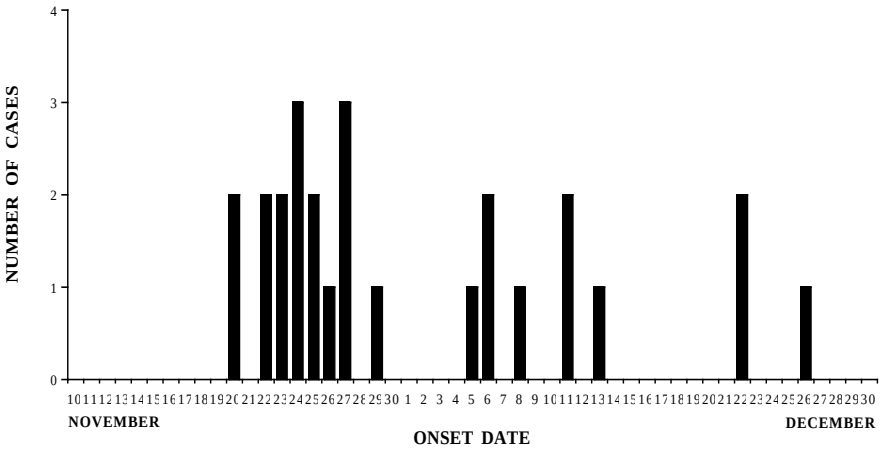


Figure 1. Onset of Confirmed *Salmonellosis*, Gideon, Missouri, Nov/Dec 1993.

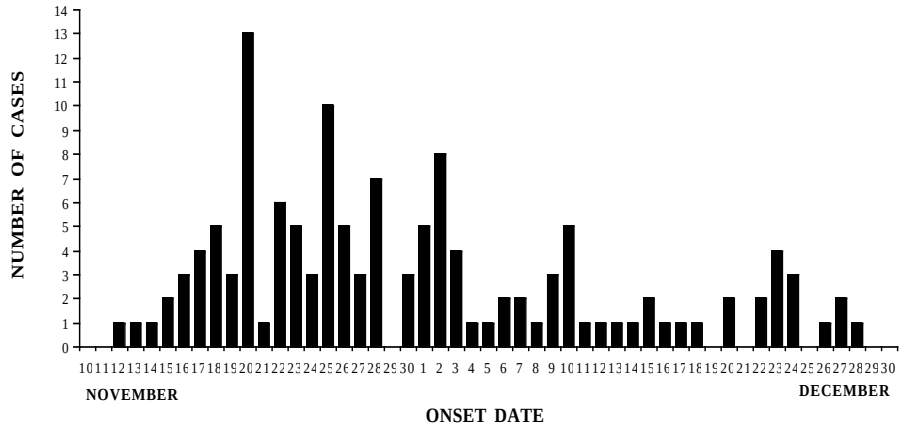


Figure 2. Onset of Diarrheal Illness, Gideon, Missouri, Nov/Dec 1993.

Community Survey

The Centers for Disease Control and Prevention (CDC) was invited to assist in the investigation and their first representatives arrived on Dec. 27. A survey of a random sample of residents of Gideon and the surrounding township was conducted by CDC and DOH. Of the 548 households in the city and township identified through personal property tax records, 150 were selected at random.

Interviews were conducted by telephone or in person. An adult family member provided information about symptoms of illness and consumption of tap water (in all forms including ice) for each member of the household. They were also questioned about their food sources and their awareness of and compliance with the boil order.

Several of the randomly selected families had moved away, so a total of 128 households were contacted and 122 (95%) agreed to be interviewed. Overall, the survey obtained information on 329 individuals, representing 20% of the 1990 census population. Diarrhea (3 or more loose stools for 2 or more days) was reported in 44% of Gideon residents in Nov. or Dec. 1993. A total of 64% of the households reported at least one member with diarrhea during that time.

All age groups were affected (median age 35 years, range 3-87 years), reflecting the census age distribution (median=35 years). The illnesses peaked during the last half of November (see Figure 2).

The attack rate for residents of the township outside Gideon who visited town for school or work was 57%. However, only 4% of township residents who did not visit Gideon between Nov. 15-30 reported illness. Persons exposed to Gideon water during that 15-day period were 10.5 times more likely to become ill than those not exposed (Relative Risk =10.5, 95% Confidence Interval =2.7-40.9).

Among Gideon residents, the amount of water consumed was a strong risk factor for diarrhea. Persons with diarrhea drank significantly more water (median=6 glasses per day) than those without diarrhea (median=4 glasses per day, probability <.01).

All but one of the Gideon households surveyed had heard about the boil order, although 20% did not hear about it until 3-11 days after it was issued. Despite knowledge of the boil order, 32% of households reported that someone in their house had drunk unboiled water after

(continued on page 4)

Reporting of Hazardous Substance Disease and Incident Encouraged by New Program

by Scott Clardy, Bureau of Environmental Epidemiologyy

The Missouri Department of Health (DOH), Bureau of Environmental Epidemiology has received a grant from the Agency for Toxic Substances and Disease Registry (ATSDR) to fund a Hazardous Substances Emergency Events Surveillance (HSEES) system.

This system was initially established by ATSDR in 1990. Currently there are 12 states participating in the HSEES system: Missouri, Rhode Island, New York, New Hampshire, Wisconsin, Iowa, Washington, Oregon, North Carolina, Texas, Alabama and Colorado.

The objectives of this system include:

1. To describe the distribution of hazardous substances emergencies within the state of Missouri;
2. To describe the morbidity and mortality experienced by responders to the event, employees, and the general public;
3. To identify the risk factors associated with the morbidity and mortality; and
4. To develop strategies to reduce subsequent morbidity and mortality.

All investigations will be documented using a hard copy ATSDR data collection form. The system will investigate non-petroleum related chemical releases. This will include agricultural chemicals, polychlorinated biphenyls (PCB's), radiological substances and other chemicals. The information to be gathered will include the event date; who the notifying agency was; event location and type (i.e. fixed facility or transportation event); weather conditions at the time of the incident; name(s) of the hazardous substance(s) involved; the type of release; quantity released; casualties involved and the extent of their injuries, how many people were exposed; if an evacuation or in-place sheltering was ordered; the actions taken to mitigate; contain; and/or control the release; and when the emergency action was terminated.

These type of incidents can lead to environmentally-induced illnesses such as acute chemical poisoning and heavy metal poisoning. The HSEES system should lead to an increased awareness of these types of events and the illnesses they can cause. This in turn should lead to increased reporting of environmentally-induced illnesses which is now required by the revised disease reporting rules (19 CSR 20-20.010 through 19 CSR 20-20.080). These rules have expanded the number and type of health conditions that must be reported to include specific environmental illnesses. Category III diseases are to be reported to DOH or the local health department within 24 hours of suspected or established diagnosis by telephone, telegraph, FAX, or other rapid communication followed by a written report within seven days.

These diseases are :

- •Acute chemical poisoning as defined in 56 FR 52166-75 (*ATSDR list of 200 hazardous substances)
- •Carbon monoxide poisoning
- •Hyperthermia
- •Hypothermia
- •Methemoglobinemia
- •Pesticide poisoning

Category IV diseases are to be reported to DOH or the local health department by written report within seven days of suspected or established diagnosis.

These diseases are:

- •Lead exposure defined as the following:
  - Blood lead level $\geq$ 10 µg/dl in persons < 18 years of age
  - Blood lead level $\geq$ 25 µg/dl in persons $\geq$ 18 years of age
- •Occupational lung disease including:
  - Silicosis
  - Asbestosis
  - Byssinosis
  - Farmer's lung
  - Toxic Organic Dust Syndrome
- •Other heavy metal poisoning including:
  - Mercury
  - Arsenic
  - Cadmium

Respiratory diseases triggered by environmental factors include environmentally or occupationally-induced asthma and bronchitis.

As with reportable communicable diseases, reporting is required from physicians, hospital and laboratories. These rules became effective in April 1993.

It is the goal of these programs to eventually discover possible relationships between different events and diseases which can be used to identify risk factors and develop prevention strategies to reduce the number of future similar events and diseases. The contact person for these programs is Scott Clardy, at (314) 751-6111 or (800) 392-7245.

* A listing of the 200 hazardous substances will be included with a letter that will be mailed to Physicians.

Food Refrigeration Advice to Prevent Food Poisoning

by David Stull, R.S., Bureau of Community Environmental Health

Inadequate cooling of food is the number one cause of foodborne illness in America today. This does not necessarily mean that our refrigeration equipment is inadequate. Sometimes it means that certain food items were left at room temperature too long before refrigerating. Other times it can mean that food was placed into containers not designed to allow food to cool rapidly, and even though the food was placed in a properly-operating refrigeration unit, the food still did not cool quickly enough to prevent rapid bacterial growth.

What foods and bacteria are involved in these foodborne illnesses?

The main foods are the one referred to as "potentially-hazardous foods." These foods are high in protein, high in moisture, and are in a form that will support the rapid and progressive growth of pathogenic bacteria. This usually (but not always) translates as red meat, pork, poultry, fish, dairy products and eggs. Sometimes vegetables, fruits and cooked grain products have been involved in outbreaks of foodborne illness. The main bacterial agents involved in foodborne illness resulting from inadequate cooling are *Salmonella, Staphylococcus, Clostridium perfringens, Shigella, Bacillus cereus* and *Yersinia enterocolitica.*

Have there been any recent foodborne outbreaks in Missouri?

Inadequate refrigeration played a role in at least five of the 36 foodborne outbreaks reported in Missouri during 1992-93. These outbreaks were caused by *Salmonella* (3), *Staph. aureus* (1), and *Campylobacter* (1), transmitted in schools, restaurants and at one catered event.

What is being done to reduce outbreaks of foodborne illnesses caused by inadequate cooling of foods, and what can you do?

Education is the key for the food-handling industry. Public health officials and the food industry have refocused inspection and training efforts to emphasize the leading causes of food borne illness. This is being done through a process called Hazardous Analysis and Critical Control Points (HACCP). The HACCP process places increased importance on the processes that control safe food product temperatures of less than 45°F or more than 140°F. Food handlers can assist by purchasing and using an accurate bayonet-type thermometer ranging from 0°F to 220°F to check temperatures of the food products. If a food product is found that has been at temperatures between 45°F and 140°F for a period of more than four hours, it should be discarded. Food products should be placed in shallow containers—no deeper than four inches — with food depth less than two and one-half inches to three inches. These containers should be left uncovered until they have cooled, stirring often. The temperature of each refrigerator should be checked to make sure it maintains an air temperature of no more than 40°F. When attempting to cool large quantities of hot foods, it is best to precool the food with an ice water bath and stir often before placing the food in the refrigerator.

The Food & Drug Administration has also recognized that the old food codes do not provide enough emphasis on proper cooling of foods and have addressed that issue in the 1993 Food Code. The new food code requires refrigerated foods to be held at 41°F (down from 45°F) and requires cooked, potentially-hazardous foods to be cooled from 140°F to 70°F within two hours and from 70°F to 41°F within four hours. Although the new FDA food code is not law in Missouri as yet, it is under consideration, and it is suggested that the new cooling temperatures be followed where possible.

Becoming aware that food in the refrigerator is not necessarily cool and understanding that active steps necessary to assist the refrigerator in doing its job, will go far toward preventing food borne illness. Food handlers must keep in mind that refrigerators are designed to keep cold foods cold and are not designed to cool hot foods.

Salmonella

(continued from page 2)
being informed of the order because they forgot (44%), did not believe there was a problem (25%), or did not fully understand that the order applied to ice and water in drinks (17%).

Since the survey sample was randomly selected and representative of the population, we can estimate that 44% of the Gideon population, or approximately 486 people, became ill during November and December with symptoms of *Salmonellosis.*

Additional Studies
The Environmental Protection Agency was invited to assist with identifying the source of the contamination. Their studies of the water distribution system, including additional testing of water and sludge samples from the fire protection tower, are still in progress.

Control Measures
DNR has carefully monitored the city's water quality since the outbreak was brought to their attention. The initial boil order was lifted on Jan. 20, 1994, after acceptable chlorine levels were demonstrated throughout the water distribution system. The private water tower was separated from the system. The city continues to use temporary chlorination equipment and is aggressively pursuing funding for a permanent system.

This report is based on information submitted by Sue Tippen, DOH Southeastern District Office, and Frederick J. Angulo, D.V.M., Ph.D., CDC. Other persons and agencies who were instrumental to the success of this investigation include: John Hill, DNR Southeast Regional Office; Microbiology and Environmental Bacteriology Units, State Public Health Laboratory; Don Sharp, M.D., CDC; Edwin E. Geldreich, EPA; and the New Madrid County Health Department.

REFERENCE
1. CDC. Waterborne disease outbreaks, 1986-1988. MMWR 1990;39:SS-1.

Isolation of *Borrelia burgdorferi* from Rabbit Ticks in Southeast Missouri

by Michael Fobbs, B.A., Bureau of Communicable Disease Control

In June of 1991 the Missouri Department of Health requested help from the Centers for Disease Control and Prevention (CDC) to determine if cases of Lyme disease reported in Missouri represented infections with *Borrelia burgdorferi* or some other phenomenon.

Since that time a series of articles in the *Missouri Epidemiologist* (January/February 1992, May/June 1992, March/April 1993) and other periodicals have commented on the question of Lyme disease in Missouri. These previous articles have discussed the feasibility of *Borrelia burgdorferi* in *Amblyomma americanum* and other ticks in Missouri, whether *Ixodes dammini* is *Ixodes scapularis*, the presence of erthema migrans(EM) or EM-like rash in Missouri and if the syndrome described in Missouri is an infectious process. All of these articles have produced questions that may support or undermine a chain of evidence leading to a conclusion.

A new link in that chain has been supplied by Dr. James H. Oliver of Georgia Southern University. Under a grant funded by the CDCs Dr. Oliver and his team have been doing ecological research on Lyme disease in Missouri. In preliminary comments about his research he shared these findings with the Missouri Department of Health.

Three isolates of a spirochete characterized as *B. burgdorferi* were cultured from ticks of the species *Ixodes dentatus*, that were feeding on two cottontail rabbits on a farm in southeast Missouri (1).

The spirochetes have been described as being atypical and different from the laboratory strain B-31 of *B. burgdorferi* and the Bibb County, Georgia isolate which was also recovered from *I. dentatus*.

Isolates reacted positively with *B. burgdorferi* monoclonal antibodies of Osp A (H3TS, H5332) and negative to Osp B (H6831), positive to Borrelia genus specific H9724 and negative to *B. hermsi* 9826. Polymerase Chain Reaction results have shown amplification of *fla* and *Osp A* genes but not of the conserved DNA sequence demonstrated by Rosa in 17 of 18 *B. burgdorferi* strains (2). One set of *Osp A* primers did not amplify the target sequence, but a different set of *Osp A* primers did amplify the target sequence.

Dr. Oliver will be attempting to culture the spirochetes from small mammals in this area and establishing a laboratory colony of *I. dentatus* to perform further tests. He will also attempt to determine if laboratory rodents can be infected by the *Ixodes dentatus* spirochete isolates via needle inoculation or by *Ixodes scapularis* or *Amblyomma americanum* tick bite.

These results are very encouraging and provide support for Lyme disease in Missouri, but do not explain how *I. dentatus* which rarely bites humans can transmit the infection to them. Dr. Oliver suspects a bridge vector which then transmits disease to humans. These findings have encouraged Dr. Oliver and others to pursue further links in the chain.

1. Oliver JH. Personal Communication, Georgia Southern University.
2. Rosa, P.A. and T.G. Schwan. 1989, *Journal of Infectious Diseases* 160:1018-1029.

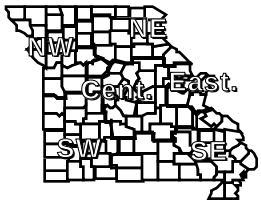
Collaborative Study on *E. coli* O157:H7

by Dr. Andrew Carson, VMD, MS, PhD, UMC and Beverley Payne, MPH, State Public Health Laboratory

The Missouri Department of Health and the WHO Collaborating Center for Enteric Zoonoses in the University of Missouri College of Veterinary Medicine are collaborating on a study to explore enhanced methods for identification and characterization of *E. coli* O157:H7. Human and animal isolates of enterohemorrhagic *E. coli* are being characterized and compared by MUG test, verocytotoxicity and hybridization with DNA toxin gene probes. Genomic "fingerprints" of *E. coli* O157:H7 isolates have been derived by pulsed field gel electrophoresis of enzymatic (Xbal) digests and random amplified polymorphic DNA (RAPD) analysis using PCR. The latter tests are particularly suitable for molecular epidemiology as they generate very specific patterns for *E. coli* serotypes, strains and isolates.

Service and research staff have discussed the feasibility of doing a regional survey of dairy farms in southwest Missouri. "Fingerprint" patterns of *E. coli* O157:H7 isolates (and several other enterohemor-rhagic serotypes) would be compared to human isolates for evidence of similarity and, therefore, zoonotic implications. Research scientists on the team have also targeted certain characteristic DNA fragments which may have potential as diagnostic probes for *E. coli* O157:H7.

Planning efforts center on targeting the most appropriate areas of direction and or focus and potential funding agencies. Subsequent issues of the *Missouri Epidemiologist* will contain updates on activities and progress. Meanwhile, we would appreciate comments and suggestions with respect to your interest in *E. coli* characterization and identification. Send comments to Dr. Andrew Carson, University of Missouri Columbia, Veterinary Microbiology, 104 Connaway Hall, 2412 Ridgefield, Columbia, MO 65203 or Beverley Payne, Missouri State Pubic Health Laboratory, 307 West McCarty Street, Jefferson City, MO 65101.



Missouri Department of Health
Disease Prevention - Communicable Disease Control
BIMONTHLY MORBIDITY REPORT

Reporting Period *
November/December 1993

	Districts							KANSAS CITY	ST. LOUIS CITY	ST. LOUIS CO.	SPGFLD GREENE CO.	2 MONTH STATE TOTALS		CUMULATIVE		
	** NW	NE	CD	SE	** SW	** ED	*** OTHER					1993	1992	FOR 1993	FOR 1992	5 YR MEDIAN
Vaccine Preventable Dis.																
Chickenpox	268	117	234	438	265	332		0	3	0	4	1661	1832	9609	10009	10009
Diphtheria	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Hib Meningitis	0	1	0	0	2	0		0	0	1	0	4	8	12	22	88
Hib Other Invasive	3	1	1	3	4	2		2	3	6	1	26	29	123	59	**
Influenza	2	2	6	1	0	1		1	3	8	1	25	49	272	111	220
Measles	0	0	0	0	0	0		0	0	0	0	0	0	1	0	65
Mumps	3	0	1	0	0	4		0	0	0	1	9	4	46	39	62
Pertussis	5	1	2	4	0	0		1	3	2	1	19	23	144	120	116
Polio	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Rubella	0	0	0	0	0	0		0	0	0	0	0	0	1	1	3
Tetanus	1	0	0	0	0	0		0	0	0	0	1	0	1	1	1
Viral Hepatitis																
A	4	1	10	5	2	23		8	54	30	11	148	472	1443	1500	810
B	4	0	4	5	4	4		9	42	8	5	85	108	585	535	633
Non A - Non B	0	0	1	0	0	0		0	0	0	1	2	4	25	27	42
Unspecified	0	0	0	0	1	0		1	0	0	0	2	0	19	9	15
Meningitis																
Aseptic	4	1	7	3	9	5		0	0	8	2	39	39	275	272	246
Meningococcal	1	0	0	0	1	0		0	0	0	1	3	12	34	32	32
Other	6	1	0	1	3	0		0	1	6	0	18	6	78	43	64
Enteric Infections																
Campylobacter	7	2	18	13	5	4		3	4	23	5	84	79	616	614	547
Salmonella	5	6	24	41	6	6		6	8	13	2	117	64	529	426	676
Shigella	5	0	7	30	1	31		3	7	14	3	101	90	674	742	411
Typhoid Fever	0	0	0	0	0	0		0	0	0	0	0	1	2	3	3
Parasitic Infections																
Amebiasis	3	0	2	2	0	0		0	1	2	0	10	0	54	23	25
Giardiasis	18	7	34	27	15	26		9	6	29	7	178	114	770	739	790
Sexually Transmitted Dis.																
AIDS	13	0	13	2	6	7	5	30	31	23	6	136	95	1664	662	599
Gonorrhea	76	31	74	90	48	22		490	800	358		1989	2378	13147	14811	17488
Genital Herpes	37	24	62	18	61	24		134	102	133		595	712	3729	3666	3244
Nongonoc. urethritis	9	4	34	28	10	0		282	589	77	9	1042	979	6425	6855	7606
Prim. & Sec. syphilis	1	0	0	2	0	3		20	122	40		188	190	1354	1170	273
Tuberculosis																
Extrapulmonary	1	1	2	0	1	1	1	0	0	2	0	9	9	45	41	42
Pulmonary	1	0	2	3	3	3	3	4	6	5	1	31	45	211	204	227
Zoonotic																
Animal Bites	68	37	49	83	52	99		0	1	289	20	698	811	6503	6751	6514
Psittacosis	0	0	0	0	0	0		0	0	0	0	0	0	1	1	1
Rabies (Animal)	0	0	2	2	0	1		0	0	0	0	5	3	35	37	36
Rocky Mtn. Sp. Fever	0	0	0	1	0	0		0	0	0	0	1	0	20	24	36
Tularemia	0	0	0	0	0	0		1	0	0	0	1	0	17	34	39

Low Frequency Diseases

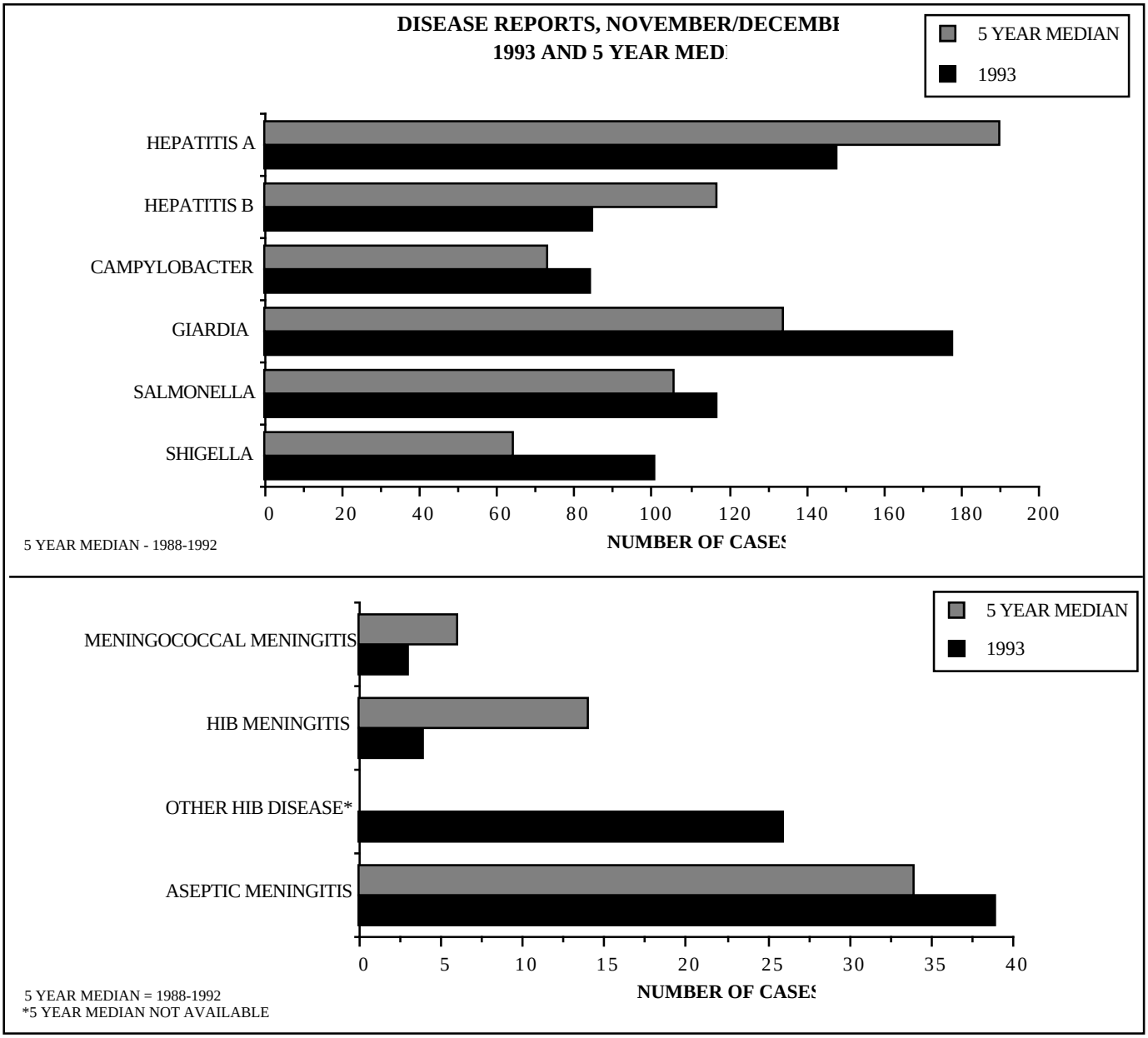
Anthrax
Botulism
Brucellosis
Chancroid
Cholera
Cryptosporidiosis
Encephalitis (infectious) - 8
Encephalitis (viral/arbo-viral)
Granuloma Inguinale
Kawasaki Disease
Legionellosis - 7
Leptospirosis
Lymphogranuloma Venereum
Malaria - 1
Plague
Rabies (human)
Reye's Syndrome
Rheumatic fever, acute
Toxic Shock Syndrome
Trichinosis

Outbreaks

Foodborne - 3
Waterborne - 1
Nosocomial - 15
Pediculosis
Scabies - 1
Other
ARI - 1
AGI - 2

*Reporting Period Beginning November 1, Ending December 31, 1993.
**Totals do not include KC, SLC, SLCo, or Springfield
***State and Federal Institutions
** Data not available

Due to data editing, totals may change.



VIRAL HEPATITIS

The number of cases of Hepatitis A continues to fall. It decreased 68.6%, from 472 cases in November/December 1992 to 148 during the same period in 1993. This is a decrease of 22.1% from the five year median of 190 cases. Hepatitis B, at 85 cases in 1993 is down 21.3% from 108 cases in 1992. It is down 27.4% from the five year median of 117 cases.

ENTERICS

Campylobacter increased 6.3%, from 79 cases in 1992 to 84 in 1993. This is an increase of 15.1% from the five year median of 73 cases. Salmonella, at 177 cases, is up 82.8% from 64 cases in 1992 and up 10.3% from the five year median of 106 cases. Shigellosis increased 12.2% from 90 cases in 1992 to 101 in 1993. It increased 57.8% from the five year median of 64 cases.

PARASITES

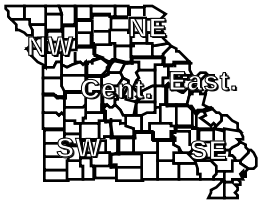
Giardiasis, at 178 cases, increased 56.1% from 114 cases in 1992. It is up 32.8% from the five year median of 134 cases.

MENINGITIS

Aseptic meningitis showed no change from 39 cases in 1992 to 39 cases in 1993. It is 14.7% above the five year median of 34 cases. Meningococcal meningitis decreased 75.0% from 12 cases in 1992 to 3 in 1993. It decreased 50.0% from the five year median of 6 cases.

HIB DISEASE

Four cases of Hib meningitis were reported for the 1993 period, a decrease of 50.0% from 8 in 1992. This is a decrease of 71.4% cases from the five year median of 14 cases. Other invasive Hib disease decreased 10.3% from 29 cases in 1992 to 26 cases in 1993. There is no five year median available for other invasive Hib disease.



Missouri Department of Health
Bureau of Communicable Disease Control
BIMONTHLY MORBIDITY REPORT

Reporting Period *
January/February 1994

	Districts							KANSAS CITY	ST. LOUIS CITY	ST. LOUIS CO.	SPGFLD GREENE CO.	2 MONTH STATE TOTALS		CUMULATIVE		
	** NW	NE	CD	SE	** SW	** ED	*** OTHER					1994	1993	FOR 1994	FOR 1993	5 YR MEDIAN
Vaccine Preventable Dis.																
Chickenpox	828	69	142	675	241	468		0	0	0	2	2425	2255	2425	2255	2255
Diphtheria	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Hib Meningitis	0	0	1	1	0	0		0	0	1	0	3	3	3	3	10
Hib Other Invasive	0	0	0	1	2	1		4	0	0	0	8	13	8	13	**
Influenza	3	14	20	12	8	10		2	1	48	13	131	141	131	141	129
Measles	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Mumps	1	0	1	0	0	1		0	0	1	0	4	5	4	5	5
Pertussis	2	0	0	0	0	0		0	0	1	0	3	10	3	10	10
Polio	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Rubella	0	0	0	0	0	0		0	0	0	0	0	1	0	1	1
Tetanus	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Viral Hepatitis																
A	2	1	1	5	5	8		1	24	22	4	73	389	73	389	108
B	3	1	1	0	7	7		2	31	4	3	59	92	59	92	78
Non A - Non B	0	0	0	0	1	1		0	0	0	1	3	6	3	6	6
Unspecified	0	0	0	0	0	0		0	0	0	0	0	3	0	3	2
Meningitis																
Aseptic	2	0	1	0	0	1		3	1	8	1	17	18	17	18	17
Meningococcal	4	0	1	1	2	4		1	0	6	1	20	5	20	5	5
Other	1	1	0	1	1	2		0	0	2	1	9	8	9	8	9
Enteric Infections																
Campylobacter	5	0	0	2	3	8		3	3	8	3	35	55	35	55	42
Salmonella	4	1	5	5	2	4		8	1	4	5	39	37	39	37	55
Shigella	2	0	0	4	0	5		0	2	1	3	17	88	17	88	39
Typhoid Fever	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Parasitic Infections																
Amebiasis	2	0	0	1	0	0		0	0	1		4	6	4	6	6
Giardiasis	10	5	3	9	7	3		7	5	13	3	65	87	65	87	81
Sexually Transmitted Dis.																
AIDS	7	2	11	3	0	6	1	34	35	15	7	121	772	121	772	83
Gonorrhea	72	14	78	55	49	19		502	393	267		1449	1811	1449	1811	2587
Genital Herpes	36	9	30	24	66	23		121	120	78		507	605	507	605	541
Nongonoc. urethritis	10	5	26	29	2	0		267	550	45	1	935	895	935	895	929
Prim. & Sec. syphilis	1	1	1	5	1	3		22	118	27	0	179	220	179	220	68
Tuberculosis																
Extrapulmonary	0	0	0	0	0	0	0	1	0	0	1	2	3	2	3	3
Pulmonary	2	0	1	7	2	0	1	3	0	2	2	20	18	20	18	20
Zoonotic																
Animal Bites	101	41	40	74	74	49		0	0	262	9	650	662	650	662	617
Psittacosis	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Rabies (Animal)	0	0	0	0	1	0		0	0	0	1	2	1	2	1	2
Rocky Mtn. Sp. Fever	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Tularemia	0	0	0	0	0	0		0	0	0	1	1	1	1	1	2

Low Frequency Diseases

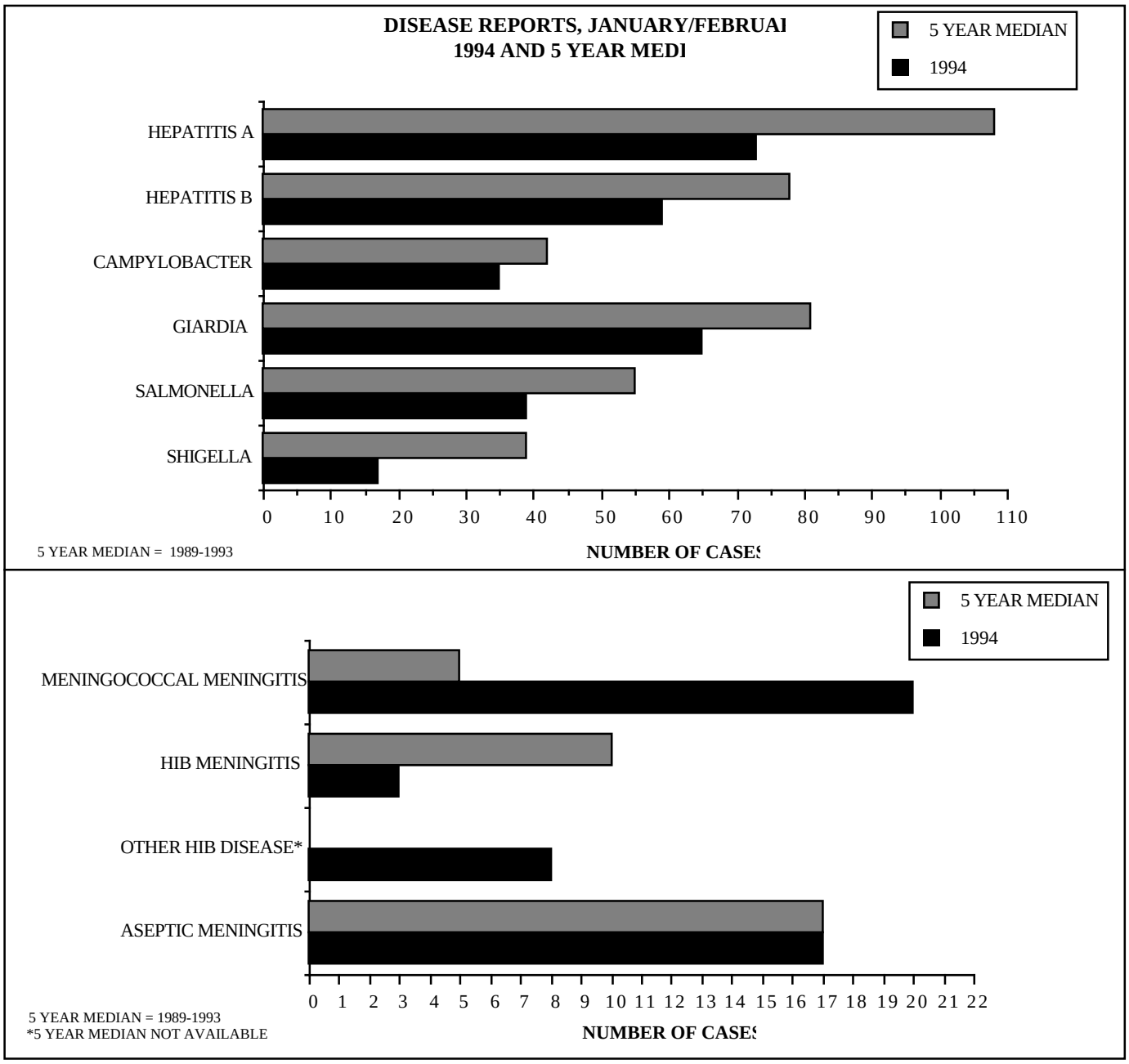
Anthrax
Botulism
Brucellosis
Chancroid
Cholera
Cryptosporidiosis
Encephalitis (infectious) - 3
Encephalitis (viral/arbo-viral)
Granuloma Inguinale
Kawasaki Disease - 1
Legionellosis - 9
Leptospirosis
Lymphogranuloma Venereum - 1
Malaria - 2
Plague
Rabies (human)
Reye's Syndrome
Rheumatic fever, acute
Toxic Shock Syndrome
Trichinosis

Outbreaks

Foodborne - 3
Waterborne
Nosocomial - 2
Pediculosis
Scabies
Other
Giardia - 1
Fifth Disease - 1
Strep Pneumonia - 1

*Reporting Period Beginning January 2, Ending February 26, 1994.
**Totals do not include KC, SLC, SLCo, or Springfield
***State and Federal Institutions
** Data not available

Due to data editing, totals may change.



VIRAL HEPATITIS

Hepatitis A decreased 82.2%, from 389 cases in January/February 1993 to 73 during the same period in 1994. It decreased 32.4% from the five year median of 108 cases. Hepatitis B decreased 35.8%, from 92 in 1993 to 59 in 1994. It is down 24.4% from the five year median of 78 cases.

ENTERICS

Campylobacter, at 35 cases, is down 36.4% from 55 cases in 1994. It decreased 16.6% from the five year median of 42 cases. Salmonella, at 39 cases, is up 5.4% from 37 cases in 1993. It is down 29.1% from the five year median of 55 cases. Shigellosis decreased 80.6% from 88 cases in 1993 to 17 in 1994. It is down 56.4% from the five year median of 39 cases.

PARASITES

Giardiasis, at 65 cases, decreased 25.3% from 87 cases in 1993. It is down 19.8% from the five year median of 81 cases.

MENINGITIS

Aseptic meningitis, at 17 cases in 1994 (also the five year median), is down 5.5% from 18 cases in 1993 . Meningococcal meningitis increased 300.0% from 5 cases in 1993 (this is also the five year median) to 20 in 1994.

HIB DISEASE

Three cases of Hib meningitis were reported during the 1994 time period, the same as reported in 1993. This is a decrease of 70.0% from the five year median of 10 cases. Other invasive Hib disease decreased 38.5% from 13 cases in 1993 to 8 in 1994. There is no five year median available for other invasive Hib disease.

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J/A	= July/August
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State Public Health Laboratory Report
Newborn Screening — Hypothyroidism, Phenylketonuria, Galactosemia and Hemoglobinopathies

James Baumgartner, B.S., M.B.A., Chief, Metabolic Disease Unit

	Nov. 93	Dec. 93	Total YTD
Specimens Tested	10,095	9,525	118,125
Initial (percent)	65.5%	64.5%	78,351
Repeat (percent)	34.5%	35.5%	39,774
Specimens: Unsatisfactory	130	107	1319
HT Borderline	886	753	8,794
HT Presumptive	54	25	328
PKU Borderline	10	25	219
PKU Presumptive Positive	1	1	14
GAL Borderline	41	25	390
GAL Presumptive Positive	2	0	39
FAS (Sickle cell trait)	85	81	1096
FAC (Hb C trait)	27	24	331
FAX (Hb variant)	23	12	176
FS (Sickle cell disease)	2	2	28
FSC (Sickle C disease)	1	0	23
FC (Hb C disease)	0	0	0

HT = Hypothyroidism, PKU = Phenylketonuria, GAL = Galactosemia, Hb = Hemoglobin, YTD = Year to Date

New Program To Determine the Number of Allergic Reactions to Flood-Related Mold

by Scott Clardy, Bureau of Environmental Epidemiology

The Missouri Department of Health's Bureau of Environmental Epidemiology will soon implement a disease surveillance system to determine the number of cases of allergic mold reactions in Missouri due to flooding. A case will be defined as any allergic reaction to mold, including sinusitis, allergic rhinitis, conjunctivitis, asthma and dermatitis. The mold can be the result of the 1993 flood or of flooding in 1994.

The bureau is seeking to obtain the prevalence rate of these conditions for several reasons. First, we want to assure proper environmental follow-up, such as appropriate disinfecting and cleaning. Second, we want to establish that patients are obtaining appropriate care for their condition. In addition, if the prevalence of flood-related allergies is significantly high, we will have cause to seek funding so that more extensive assistance may be provided. With the help of this surveillance activity we will be able to characterize those areas most affected by mold from flooding and then develop appropriate plans for the future.

We will conduct active surveillance of selected allergists and pulmonary physicians located in flood-prone areas and/or those who are likely to get referrals from such areas. We will phone these physicians to establish a contact person in each office then conduct a bi-weekly follow-up to obtain the number of cases seen in that office for the previous two weeks. We will also ask for demographic data for each case reported. Any physicians not contacted by phone, but seeing conditions which fit the case definition, are urged to report them to Scott Clardy or Daryl Roberts at (800) 392-7245.

Perinatal Hepatitis B Screening and Immunization

by Paula Rosenberg, Bureau of Immunization

During the 1993 legislative session the Missouri General Assembly passed House Bill 522 which was subsequently signed into law by Gov. Carnahan. This bill requires, among other issues, prenatal hepatitis B screening, and the immunization of newborns whose mothers are found to be infected with hepatitis B virus (HBV).

The initial panel of prenatal screening tests should now include, with the consent of the woman, a test for hepatitis B surface antigen (HBsAg) in addition to the previously required syphilis serology. The Missouri Department of Health (DOH) continues to recommend, but does not require, a test for rubella immunity. Because an epidemic of syphilis is occurring in certain areas of the state, DOH is also recommending that testing for syphilis be repeated on each pregnant woman during the second trimester, and again when the woman presents for delivery. Finally, DOH recommends that a pregnant woman at high risk for hepatitis B infection be screened again for HBsAg during the third trimester.

House Bill 522 revises 210.030 RSMo. to require those who provide prenatal care for women to screen, with the consent of the woman, for syphilis and hepatitis B using approved serological tests. These tests must be performed either at the time of the patient's first prenatal examination, or no later than twenty (20) days thereafter. If the patient refuses, the test is not required, although the clinician should explain the benefits of testing to the woman, and try to persuade her to be tested.

The revised statute also requires the obstetrical professional in attendance at the delivery of a child whose mother is infected with hepatitis B to administer the appropriate doses of hepatitis B vaccine and hepatitis B immune globulin (HBIG) within 12 hours of birth. However, the obstetrical professional may designate another person, such as a pediatrician, to administer, or order the administration of, appropriate vaccine and immunoglobulin. This would allow continuation of the prevailing medical practice in many hospitals, which is for the pediatrician or other physician caring for the newborn to administer, or provide orders for, the immunizations. It must be remembered that the law, as written, makes the obstetrical professional responsible for the appropriate immunization of the infant. This provides assurance that, even in cases where no other physician is involved, the vaccine and HBIG will be properly administered.

The revisions contained in H B 522 do not affect the requirement for syphilis screening, which remains as it was previously. There is language in 210.030 RSMo which states that prenatal testing shall also include "...such other treatable diseases and metabolic disorders as are prescribed by the department of health...." This gives DOH the authority to require testing for conditions other than syphilis and hepatitis B if it is determined, in the future, that such additional testing is necessary. As noted above, DOH currently recommends, not requires, prenatal testing for rubella and additional testing for syphilis.

Preventing perinatal HBV transmission is of critical importance due to the likelihood of chronic infection resulting from exposure early in life. According to the Immunization Practices Advisory Committee (ACIP), the risk of chronic infection is age dependent. "The risk of HBV infection among infants born to HBV-infected mothers ranges from 10% to 85%. ..Infants who become infected by transmission have a 90% risk of chronic infection, and up to 25% will die of chronic liver disease as adults"[1] With the identification of HBsAg-positive pregnant women, more than 90% of their infants can be protected if given hepatitis B vaccine and HBIG in a timely manner.

All positive prenatal HBsAg results are now reportable. The health care provider and laboratory director are both responsible for reporting to the local health agency where the pregnant women resides, or to DOH, by telephone or written report within seven (7) days of the positive test result.

When a positive test result is reported, the local health authority will work with the provider to screen and vaccinate, as appropriate, all household, needle-sharing, and sexual contacts to the positive mother. The State of Missouri, through the local health agency, will provide screening, HBIG and hepatitis B vaccine at no charge for these contacts. HBIG and hepatitis B vaccine will also be provided to the delivery hospital in order to provide protection to the infant at the time of birth.

Another major strategy for preventing hepatitis B is the integration of hepatitis B vaccine into the routine childhood immunization schedule as recommended by the American Academy of Pediatrics[2] and the ACIP[3]. Hepatitis B vaccine is now recommended for all infants and children during routine primary health care visits. DOH endorses these recommendations and is supporting them through the provision of hepatitis B vaccine to local health agencies for administration to all infants born on or after January 1, 1991.

You can obtain more information on these programs by contacting your District Immunization Representative or the Bureau of Immunization at (314) 751-6133.

1. Immunization Practices Advisory Committee (ACIP), "Hepatitis B Virus: A Comprehensive Strategy for Eliminating Transmission in the United States Through Universal Childhood Vaccination," *Morbidity and Mortality Weekly Report,* Vol. 40, No. RR-13, p.1-25, November 22, 1991.
2. Policy Statement on Universal Hepatitis B Immunization, American Academy of Pediatrics, February 1992.

Reorganization of the Bureau of AIDS Prevention and of Sexually Transmitted Diseases

by Bill Huber, Chief, Bureau of STD/HIV Prevention

There have been some internal changes in recent months within the Department of Health which affect the AIDS/HIV Program. The first step was to reorganize the Bureau of AIDS Prevention (BAP) and the Bureau of Sexually Transmitted Diseases (BSTD) into a Bureau of STD/HIV Prevention and a Bureau of HIV/AIDS Services. This reorganization was made in order to strengthen the prevention activities of BSTD and BAP.

Subsequently, the Bureau of HIV/AIDS Services was organizationally moved to the Bureau of Special Health Care Needs (BSHCN) in the Division of Maternal, Child and Family Health. This move should provide more stability for the HIV/AIDS Services program and make payment for services more efficient through the use of the established BSHCN's MOCARES information system.

Les Hancock, Chief, BSHCN now supervises the HIV/AIDS Services program and can be reached at (314) 751-6246. Bill Huber, Chief, BSTD/HIVP now supervises the STD and HIV Prevention activities. He can be reached at (314) 751-6139.

Hilda Chaski has been appointed as the departments' AIDS Coordinator to represent Dr. Coleen Kivlahan, the Director of the Missouri Department of Health, on policy issues that transcend the individual programmatic considerations. She will also assist in facilitating communication and coordination internally between divisions and externally with local public health agencies, community based organizations, and HIV/AIDS organizations. She can be reached at (314) 751-6079.

ACIP Changes in Childhood Immunization Schedule

Mary Ann Harder, Bureau of Immunization

The Advisory Committee on Immunization Practices (ACIP) of the U.S. Public Health Service recently revised the immunization schedule for infants and children. The new recommendation include two changes in the childhood immunization schedule as follows:

Measles, mumps and rubella (MMR) vaccine is recommended to be administered routinely to all children at 12-15 months of age.

The third dose of oral polio vaccine is recommended to be administered routinely at 6 months of age rather than at age 15 months along with the third doses of DTP vaccine and *Haemophilus influenzae* type b vaccine (if indicated).

Previously, the first dose of MMR was recommended at 12 months of age only in counties defined as "high risk," and 15 months elsewhere in the U.S. The major reasons for liberalizing the recommended age are:

1) data indicating that children born in the United States today receive less antibody from their mothers at birth and will respond to measles vaccine at a younger age than in the past;
2) the opportunity to increase coverage with MMR by allowing the first dose to be given as early as 12 month of age.

Available data may show that there may still be small differences between response to measles vaccine given at 12 and 15 months of age. Based on data from several recent studies, about 93% of children responded to measles vaccination at age 12 months compared with 97-99% at age 15 months. However, the second dose of measles vaccine, recommended at school entry, reduces the importance of this small difference, which may be more than balanced by improvements in coverage. Since almost all persons who do not respond to the first dose will respond to the second dose, overall immunity levels for measles will remain high in the school-age population.

The recommendation to move the third OPV to six months was based on the following:

1) administering the third OPV at 6 months induces a similar immune response to that observed when the third OPV is given at 15-18 months of age;
2) the change simplifies the immunization schedule in the first year of life because DTP, Hib, and OPV each can now be given at 2, 4, and 6 months;
3) the change should increase vaccination coverage with the third OPV.

The fourth dose of OPV is still recommended to be between 4-6 years of age and the minimum interval between OPV doses remains at 6 weeks.

The American Academy of Pediatrics Committee on Infectious Diseases (Red Book Committee) has also approved liberalizing MMR to 12-15 months of age and changed its recommendation to allow administration of the third OPV at any time from 6 through 18 months of age.

It should also be noted that the booster dose of *Haemophilus Influenzae* type B PRP-D (ProHibit) may be administered at 12 months of age following completion of the primary series, regardless of the vaccine used in the primary series.

The Missouri Department of Health endorses these schedule changes and encourages their routine implementation. Questions or concerns regarding the revised immunization schedule may be directed to your district immunization representative or to the Bureau of Immunization at (314) 751-6133.

Congenital Syphilis in Missouri

by Dr. Robert Hamm, Office of Epidemiology

During the past 4 years there has been a dramatic increase in the number of reported cases of congenital syphilis in Missouri, paralleling the increase that has occurred in cases of primary and secondary (P&S) syphilis in the state. Congenital syphilis, which is a preventable disease, causes significant fetal mortality, and it can cause serious illness, long-term sequelae, and occasional deaths in affected infants and children.

Epidemiology of Congenital Syphilis in Missouri

The annual reported incidence of congenital syphilis in Missouri from 1983 to 1993 is shown in Figure 1. Since 1989, yearly increases have occurred in the number of reported cases. The increase was especially striking between 1992 and 1993, when the annual number of cases rose from 28 to 97, an increase of 246%.

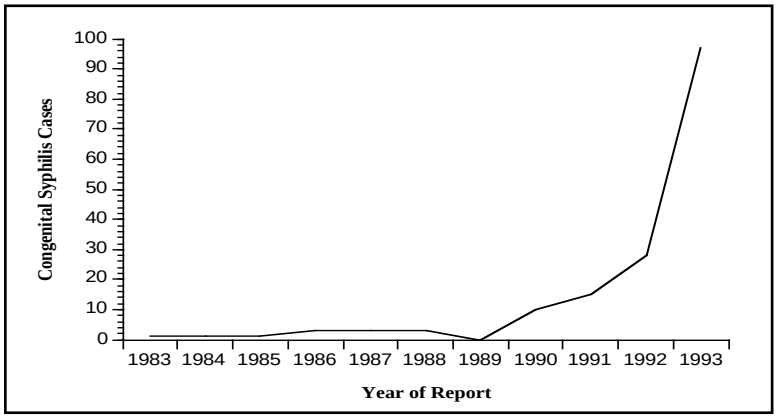


Figure 1. Congenital Syphilis Cases by Year of Report, Missouri, 1983-1993

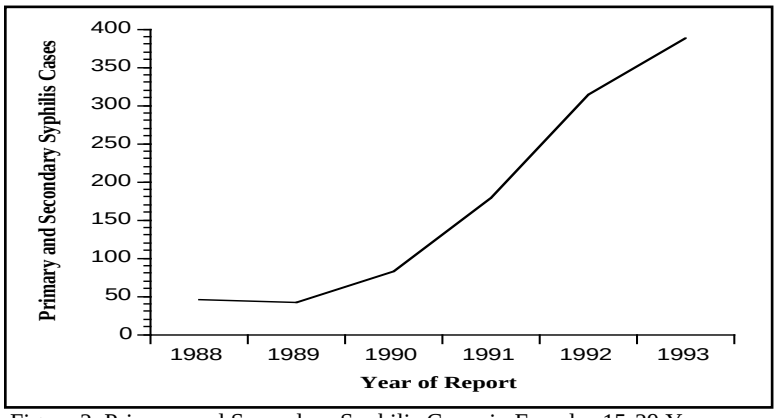


Figure 2. Primary and Secondary Syphilis Cases in Females 15-29 Years of Age by Year of Report, Missouri, 1988-1993

In interpreting these data, it must be kept in mind that a new surveillance case definition came into use in the second half of 1990. This new case definition is more sensitive than the previous case definition in that it requires the reporting of all infants whose mothers had untreated or inadequately treated syphilis at delivery, regardless of the presence of clinical signs in the infant. As a result, part of the increase in congenital cases beginning in 1990 is due to this case definition change; however, the growth in cases also reflects a true increase in the occurrence of infection in infants.[1]

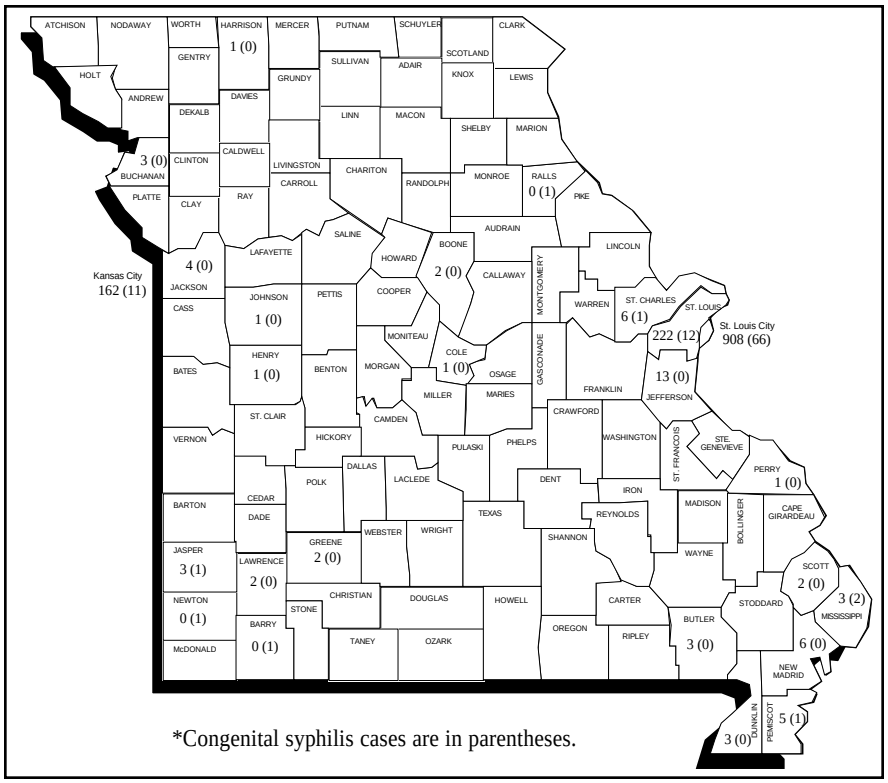
The incidence of congenital syphilis closely follows the incidence of P&S syphilis in women in the peak childbearing years. This relationship is seen when one compares the incidence curve for congenital syphilis (Figure 1) with the 1988-1993 incidence curve for P&S syphilis in women 15-29-years-of-age (Figure 2).

In Missouri, congenital syphilis cases have primarily been concentrated in the metropolitan areas of St. Louis and Kansas City, and especially during the past year in the St. Louis area (Figure 3). This corresponds to the concentration of P&S syphilis cases in these same locations.

Table 1 provides information on selected variables associated with congenital syphilis cases reported in the state during 1993. The racial composition of these cases indicates a very disproportionate representation of blacks. Eighty-six (88.7%) of the 97 congenital syphilis cases reported during 1993 were in blacks. The importance of teenage mothers in the current outbreak of congenital syphilis is reflected in the fact that the largest number of cases, 29 (29.9%), were born to mothers in the 15-19-year-old age group.

The great majority of mothers of infants with congenital syphilis were either single or separated/divorced; together these two categories accounted for 89 (91.8%) of the 97 reported cases. Lack of adequate prenatal care, which is the leading factor accounting for the failure to prevent congenital syphilis[2], was associated with a significant number of Missouri cases. During 1993, 29 (29.9%) of the 97 mothers of congenital syphilis cases received no prenatal care. The prenatal care status of 3 (3.1%) of the mothers was unknown. Of the remaining 65 mothers, who reported at least 1 prenatal clinic visit, a total of 20 (30.8%) had 3 or fewer visits; and a total of 40 (61.5%) had 6 or less visits.

In Missouri, as elsewhere, syphilis cases have occurred most frequently in persons who live in lower socioeconomic neighborhoods. In St. Louis City, for example, the median family income, based on 1990 census data, for the five



*Congenital syphilis cases are in parentheses.

Figure 3. Primary and Secondary Syphilis Cases and Congenital Syphilis Cases* by County of Residence, Missouri, 1993

SELECTED VARIABLES	MISSOURI CASES	%	ST. LOUIS CITY CASES	%	ST. LOUIS COUNTY CASES	%	KANSAS CITY CASES	%	OUTSTATE MISSOURI* CASES	%
RACE										
WHITE	8	8.2%	4	6.1%	0	0.0%	1	9.1%	3	37.5%
BLACK	86	88.7%	61	92.4%	12	100.0%	9	81.8%	4	50.0%
OTHER	3	3.1%	1	1.5%	0	0.0%	1	9.1%	1	12.5%
AGE GROUP OF MOTHER										
10-14	1	1.0%	1	1.5%	0	0.0%	0	0.0%	0	0.0%
15-19	29	29.9%	19	28.8%	3	25.0%	4	36.4%	3	37.5%
20-24	19	19.6%	14	21.2%	4	33.3%	0	0.0%	1	12.5%
25-29	27	27.8%	18	27.3%	3	25.0%	4	36.4%	2	25.0%
30-34	16	16.5%	11	16.7%	2	16.7%	1	9.1%	2	25.0%
35-44	5	5.2%	3	4.5%	0	0.0%	2	18.2%	0	0.0%
MARITAL STATUS OF MOTHER										
SINGLE (NEVER MARRIED)	84	86.6%	61	92.4%	11	91.7%	7	63.6%	5	62.5%
SEPARATED/DIVORCED	5	5.2%	3	4.5%	0	0.0%	1	9.1%	1	12.5%
MARRIED	6	6.2%	2	3.0%	0	0.0%	3	27.3%	1	12.5%
OTHER/UNKNOWN	2	2.1%	0	0.0%	1	8.3%	0	0.0%	1	12.5%
PRENATAL CARE										
YES **	65	67.0%	41	62.1%	8	66.7%	8	72.7%	8	100.0%
NO	29	29.9%	24	36.4%	2	16.7%	3	27.3%	0	0.0%
UNKNOWN	3	3.1%	1	1.5%	2	16.7%	0	0.0%	0	0.0%
TOTAL CASES	97	100.0%	66	100.0%	12	100.0%	11	100.0%	8	100.0%

*All of Missouri except St. Louis City, St. Louis County, and Kansas City
** Defined as one or more prenatal visits to a health care provider

Table 1. Selected Variables Associated With Congenital Syphilis Cases, Missouri, 1993

zip code areas with the highest rates of syphilis ranged from $10,243 to $20,211. This compares with a median family income for St. Louis of $24,274, and a statewide figure of $31,838.

An additional factor which has been related to the occurrence of congenital syphilis is the use of illicit drugs, especially crack cocaine. Crack use has been associated with high risk sexual behaviors, and with the acquisition of syphilis. It has also been associated with lack of prenatal care among pregnant female users.[3,4] While precise data on the relationship between illicit drug use and syphilis infection in Missouri is not available, there is substantial anecdotal evidence obtained by public health investigators which indicates that drugs, and especially crack cocaine, do play a significant role in promoting the spread of the disease in the state.

Transmission

Transmission of *Treponema pallidum* from mother to fetus can occur across the placenta during the prenatal period, and also at the time of delivery through contact with infectious secretions in the birth canal. Infection of the child is most likely during early maternal syphilis, with the probability of transmission being 70-100% during the first 4 years after the mother acquires her infection. Subsequently, the likelihood of transmission decreases, although it can occur throughout the latent period.[1,5]

Transmission can occur from a child with early congenital syphilis to those with whom he or she has direct contact. The moist secretions found in early congenital syphilis are highly contagious.[6]

Clinical Manifestations of Congenital Syphilis

An infant or child with congenital syphilis can present in the following ways:

- Stillbirth
- No clinical signs of disease
- Signs of early congenital syphilis (Table 2), which appear before the age of 2 years
- Signs of late congenital syphilis (Table 3), which appear after the first 2 years of life

At least half of infected liveborn infants do not have clinical evidence of disease at the time of birth (and if the mother acquired her syphilis infection late in pregnancy, she may not show any signs of disease before the time of delivery). Infants who develop signs of early congenital syphilis usually do so within the first few months after birth.[1]

If the mother has untreated early syphilis, up to 40% of pregnancies will result in stillbirths or perinatal deaths.[1] Any woman who delivers a stillborn infant after 20 weeks gestation should be tested for evidence of syphilis.[7] Stillbirths associated with syphilis should be reported to public health authorities in the same manner as any other case of congenital syphilis.

Diagnostic Considerations

The clinician can diagnose congenital syphilis with confidence when the results of dark-field microscopy or direct immunofluorescence on specimens from sites such as skin lesions, umbilicus, or placenta are positive, or when the mother's treponemal and nontreponemal tests are reactive and the infant has classic signs of disease.[1] However, the situation is usually less clear, and the presumptive diagnosis, as well as treatment decisions, must be based on:

- assessment of the mother's past and present clinical status and serologic test results, and her treatment history; and
- appropriate evaluation of the infant.

Health care providers who care for pregnant women are required by Missouri law to obtain, with the consent of the patient, a serologic test for syphilis on each pregnant woman at, or within 20 days of, her first visit for prenatal care. However, because of the congenital syphilis outbreaks that have been occurring in the state, the Missouri Department of Health currently recommends that syphilis testing be repeated on all pregnant women during the second trimester and again when the mother presents for delivery. Ideally, the results of the latter test should be known before the infant leaves the hospital. It should be noted that maternal blood is preferable to cord blood for syphilis screening at the time of birth.[1]

Any woman who is diagnosed with syphilis and/or has a child diagnosed with congenital syphilis should be tested for evidence of HIV infection unless she is already known to be infected with HIV. In areas with high HIV prevalence, any person with primary syphilis whose initial HIV test is negative should be retested for HIV after 3 months.[7] Prompt diagnosis and treatment of syphilis (and other sexually transmitted diseases) may decrease the overall risk of HIV transmission in the community since the presence of a disease such as syphilis significantly enhances the efficiency of HIV transmission.[8]

Osteochondritis	Jaundice
Snuffles	Pseudoparalysis
Rash	Lymphadenopathy
Condyloma lata	Mucous patches
Anemia	Edema
Hepatosplenomegaly	

Table 2. Clinical Signs of Early Congenital

Frontal bosses	Hutchinson's incisors
Short maxillas	Mulberry molars
Saddle nose	High palatal arch
Protruding mandible	Saber shins
Interstitial keratitis	Flaring scapulas
Eighth nerve deafness	Sternoclavicular thickening
Clutton's joints	

Table 3. Clinical Signs of Late Congenital Syphilis

In general, a seropositive pregnant woman should be considered infected unless a history of adequate treatment is clearly documented in her medical record and sequential serologic antibody titers have appropriately declined. Table 4 describes infants who should have a specific evaluation for congenital syphilis based on the mother's serologic test results and her history of treatment and follow-up. Infants who require evaluation for congenital syphilis should undergo the examinations and tests listed in Table 5.[7]

Serologic test results are usually the basis for diagnosing syphilis, and they are the screening tools used to identify potential congenital syphilis patients. However, these tests require careful interpretation. Two important points to be remembered are: First, if maternal infection occurs late in pregnancy, the infected infant (and possibly also the mother) can have a negative serologic test for syphilis at the time of birth, and the infant (and possibly also the mother) may have no signs of syphilis infection. In this situation, the infant can remain seronegative until after 1 month of age. Second, a mother appropriately treated for syphilis during pregnancy can still passively transfer nontreponemal and treponemal antibodies to the fetus, and at the time of birth, the infant, though uninfected, will have positive nontreponemal and treponemal antibody tests. The nontreponemal titer will usually be less than or equal to the corresponding titer in the mother, and it should revert to negative in approximately 4-6 months.[1,6]

Treatment Considerations

The CDC has recently issued comprehensive guidelines for the management and treatment of syphilis and other sexually transmitted diseases.[7] The following recommendations are taken directly from these guidelines.

A. Pregnant women with syphilis

Treatment during pregnancy should be the **penicillin regimen** appropriate for the woman's stage of syphilis. Some experts recommend additional therapy (e.g., a second dose of **benzathine penicillin** 2.4 million units IM) 1 week after the initial dose, particularly for those women in the third trimester of pregnancy and for women who have secondary syphilis during pregnancy.

Following treatment, serologic titers should be checked monthly until adequacy of treatment has been assured. The antibody response should be appropriate for the stage of disease.

Women who are treated for syphilis during the second half of pregnancy are at risk for premature labor or fetal distress, or both, if their treatment precipitates the Jarisch-Herxheimer reaction. These women should be advised to seek medical attention following treatment if they notice any change in fetal move-

ments or if they have contractions. Stillbirth is a rare complication of treatment; however, since therapy is necessary to prevent further fetal damage, that concern should not delay treatment.

There are no proven alternatives to penicillin. A pregnant woman with a history of penicillin allergy should be treated with penicillin, after desensitization, if necessary. Skin testing may be helpful for some patients and in some settings. CDC has developed a detailed set of recommendations[7] for management of patients with a history of penicillin allergy.

Tetracycline and doxycycline are contraindicated during pregnancy. Erythromycin should not be used because it cannot be relied upon to cure an infected fetus.

B. Congenital syphilis in infants during the newborn period

Infants who meet any of the criteria listed in Table 6 should be treated for presumed congenital syphilis . Recommended treatment regimens for infants are:

Aqueous crystalline penicillin G, 100,000-150,000 units/kg/day (administered as 50,000 units/kg IV every 12 hours during the first 7 days of life and every 8 hours thereafter) for 10-14 days,

or

Procaine penicillin G, 50,000 units/kg IM daily in a single dose for 10-14 days.

If more than one day of therapy is missed, the entire course should be restarted. Note that infants with clinically evident congenital syphilis should have an ophthal-mologic exam.

One situation which may arise involves an infant whose complete evaluation for congenital syphilis (see Table 5) was normal and whose mother was either:

a) treated for syphilis during pregnancy with erythromycin; or
b) treated for syphilis <1 month before delivery; or

Infants should be evaluated for congenital syphilis if they were born to seropositive (nontreponemal test confirmed by treponemal test) women who meet the following criteria:

- Have untreated syphilis*; or
- Were treated for syphilis during pregnancy with erythromycin [or any non-penicillin antibiotic]; or
- Were treated for syphilis <1 month before delivery; or
- Were treated for syphilis during pregnancy with the appropriate penicillin regimen, but nontreponemal antibody titers did not decrease sufficiently after therapy to indicate an adequate response (≥ fourfold decrease); or
- Do not have a well-documented history of treatment for syphilis; or
- Were treated appropriately before pregnancy but had insufficient serologic follow-up to assure that they had responded appropriately to treatment and are not currently infected (≥ fourfold decrease for patients treated for early syphilis; stable or declining titers ≤1:4 for other patients).

* A woman treated with a regimen other than those recommended for treatment of syphilis (for pregnant women or otherwise) in the current treatment guidelines[7] should be considered untreated.

Table 4. Infants Who Should Be Evaluated for Congenital Syphilis Based on the Mother's Test Results, Treatment History, and History of Follow-Up[7]

The clinical and laboratory evaluation of infants for congenital syphilis should include the following:

- A thorough physical examination for evidence of congenital syphilis;
- A quantitative nontreponemal serologic test for syphilis performed on the infant's sera (not on cord blood);
- CSF analysis for cells, protein, and VDRL;
- Long bone x-rays;
- Other tests as clinically indicated (e.g., chest x-ray, complete blood count, differential and platelet count, liver function tests);
- For infants who have no evidence of congenital syphilis on the above evaluation, determination of presence of specific antitreponemal IgM antibody by a testing method recognized by CDC as having either provisional or standard status;
- Pathologic examination of the placenta or amniotic cord using specific fluorescent antitreponemal antibody staining.

Table 5. Clinical, Laboratory, and X-Ray Evaluation of Infants for Congenital Syphilis[7]

Infants should be treated for presumed congenital syphilis if they were born to mothers who, at delivery, had untreated syphilis* or who had evidence of relapse or reinfection after treatment. Additional criteria for presumptively treating infants for congenital syphilis are as follows:

- Physical evidence of active disease;
- X-ray evidence of active disease;
- A reactive CSF-VDRL or, for infants born to seroreactive mothers, an abnormal CSF white blood cell count or protein, regardless of CSF serology;
- A serum quantitative nontreponemal serologic titer that is at least fourfold greater than the mother's titer**;
- Specific antitreponemal IgM antibody detected by a testing method that has been given provisional or standard status by CDC;
- If they meet any of the criteria listed in Table 4 but have not been fully evaluated.

* A woman treated with a regimen other than those recommended for treatment of syphilis (for pregnant women or otherwise) in the current treatment guidelines[7] should be considered untreated.

** The absence of a fourfold greater titer for an infant cannot be used as evidence against congenital syphilis.

Table 6. Infants Who Should Be Treated for Presumed Congenital Syphilis[7]

c) treated with an appropriate regimen before or during pregnancy but did not yet have an adequate serologic response.

In this case, the infant should be treated with **benzathine penicillin G,** 50,000 units/kg IM in a single dose. In some instances, infants with a normal complete evaluation for whom follow-up can be assured can be followed closely without treatment.

C. Older infants and children with congenital syphilis

After the newborn period, children diagnosed with syphilis should have a CSF examination to exclude neurosyphilis and records should be reviewed to assess whether the child has congenital or acquired syphilis. Any child who is thought to have congenital syphilis (or who has neurologic involvement) should be treated with **aqueous crystalline penicillin G**, 200,000-300,000 units/kg/day IV or IM (administered as 50,000 units/kg every 4-6 hours) for 10-14 days.

D. Guidelines for follow-up evaluation of treated and non-treated infants and children

1. Infants treated for congenital syphilis during the newborn period.

Treated infants should be followed every 2-3 months to assure that nontreponemal antibody titers decline; these infants should become nonreactive by 6 months of age. Treponemal tests should not be used to evaluate response to treatment because test results can remain positive despite effective therapy if the child was infected. Infants with CSF pleocytosis should undergo CSF examination every 6 months, or until the cell count is normal. If the cell count is still abnormal after 2 years, or if a downward trend is not present at each examination, the child should be re-treated. The CSF-VDRL also should be checked at 6 months; if still reactive, the infant should be re-treated.

2. Children treated for congenital syphilis after the newborn period.

Follow-up of these children should be the same as that prescribed for congenital syphilis among neonates. Nontreponemal antibody titer decline may be slower than in infants treated during the neonatal period.

3. Seroreactive infants (or infants whose mothers were seroreactive at delivery) who were not treated for congenital syphilis during the perinatal period.

These infants should receive careful follow-up examinations at 1, 2, 3, 6, and 12 months after birth. Nontreponemal antibody titers should decline by 3 months of age and should be nonreactive by 6 months of age if the infant was not infected and the titers were the result of passive transfer of maternal antibody. If these titers are found to be stable or increasing, the child should be re-evaluated, including CSF examination, and fully treated. Passively transferred treponemal antibodies may be present for as long as 1 year. If they are present >1 year, the infant should be re-evaluated and treated for congenital syphilis.

Prevention

Congenital syphilis is, as mentioned above, a preventable disease. One very important step in preventing congenital syphilis is to ensure that all pregnant women receive appropriate prenatal care, which is started early in pregnancy, and which includes clinical and serologic evaluation for evidence of syphilis. If syphilis is detected, the woman must receive adequate treatment, and then appropriate follow-up to ensure that the treatment was successful.

A second important means of preventing congenital syphilis is to identify and appropriately treat cases of primary and secondary syphilis, and to assure that contacts of these cases are identified, evaluated, and treated. This will result in lower levels of syphilis in the community, and thus will lessen the probabilities that infection will occur in pregnant women. Physicians and other health care providers play a vital role in this process through the diagnosis and treatment of syphilis in their patients, and through the prompt reporting of these patients to public health authorities. Such reporting, in addition to being required by Missouri law, is very important in helping to assure that the sexual partners of these cases will be identified, properly evaluated, and treated as necessary.

Although syphilis cases in Missouri have been concentrated in the metropolitan areas, they can occur anywhere, including small rural counties. Consequently, clinicians need to maintain an appropriate index of suspicion for syphilis (and other sexually transmitted diseases) in their patients. A risk assessment for sexually transmitted diseases should be performed on every patient, along with clinical and laboratory evaluation as necessary. Persons with a sexually transmitted disease, or who are at risk based on their current behavior, can benefit from education and ongoing counseling to help them eliminate or reduce their risk of subsequent infection. Finally, persons treated for syphilis need to have adequate follow-up to ensure that treatment was effective and that reinfection has not occurred.

References

1. Zenker PN, Berman SM. Congenital syphilis: trends and recommendations for evaluation and management. *Pediatr Infect Dis J* 1991; 10:516-22.

2. Schulz KF, Murphy FK, Patamasucon P, Meheus AZ. Congenital syphilis, in Holmes KK, Mardh PA, Sparling PF, Wiesner PJ (eds.). *Sexually Transmitted Diseases* (2nd Ed.). New York: McGraw-Hill, 1990; pp. 821-842.

3. Marx R, Aral SO, Rolfs RT, et al. Crack, sex, and STD. *Sex Transm Dis* 1991; 18:92-101.

4. CDC. Guidelines for the prevention and control of congenital syphilis. *MMWR* 1988; 37(no. S-1):1-13.

5. Benenson AS. *Control of Communicable Diseases in Man*. Washington: American Public Health Association, 1990; pp.420-425.

6. Committee on Infectious Diseases. *Report of the Committee on Infectious Diseases (22nd Ed.)*. Elk Grove Village, IL: American Academy of Pediatrics, 1991; pp.453-62.

7. CDC. 1993 Sexually transmitted diseases treatment guidelines. *MMWR* 1993; 42(No. RR-14):27-46.

8. Wasserheit JN. Epidemiological synergy. *Sex Transm Dis* 1992; 19:61-77.

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Missouri EPIDEMIOLOGIST

Volume XVI, Number 3 *May–July 1994*

Bureau of Communicable Disease Control 1993 Annual Report

Michael Fobbs, B.A.
Mahree Fuller Skala, M.A.
Bureau of Communicable Disease Control

Enteric Diseases

There were 35 *E. coli* O157:H7 cases reported during 1993, the first complete year of reporting for this disease. The rates per 100,000 population reported in the Eastern and Southwestern districts are of particular interest because they are more than double the rates in the other districts. See Figure 1. There is still significant under-detection and under-reporting of this pathogen, which prospective studies in other states have found to be more common than *Shigella*.[1]

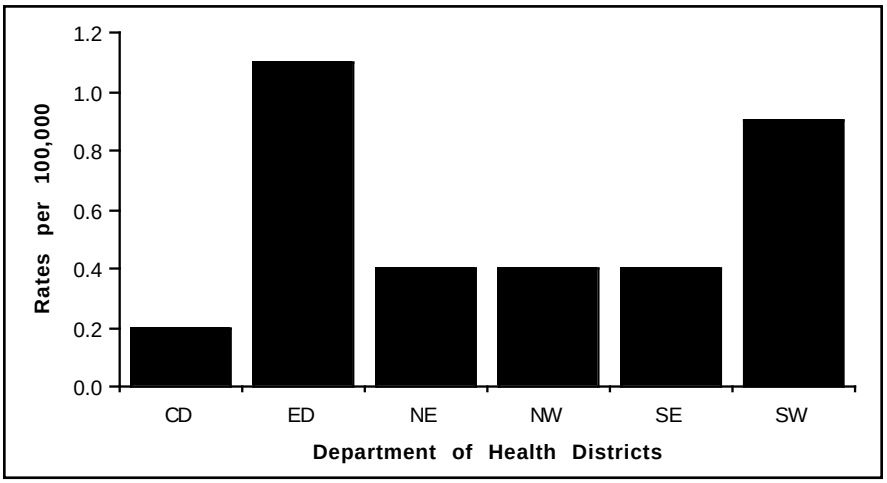

Figure 1. *E. coli* O157:H7 incidence rates per 100,000 by Department of Health district, Missouri, 1993.

Shigellosis was hyper-endemic in some urban areas of the state in 1992, with a total of 742 cases being reported. In 1993, the reported incidence of this disease decreased by 9.2 percent to 674 cases. Reductions in the number of reported cases were seen in the Eastern,

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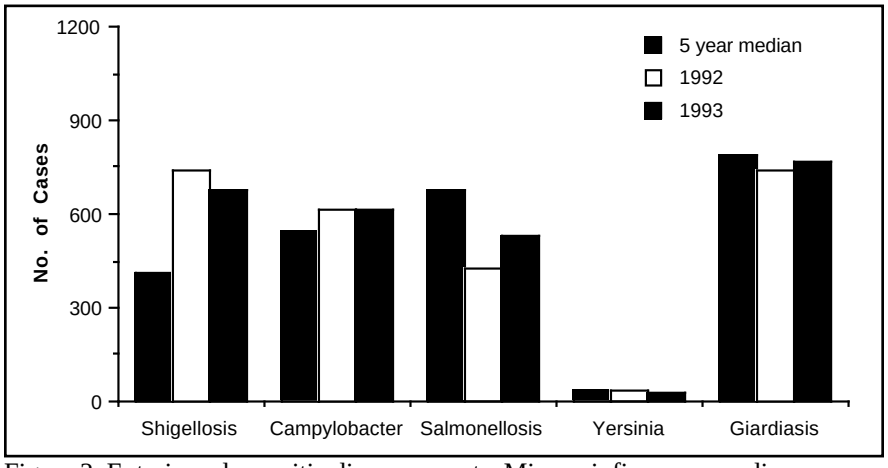

Figure 2. Enteric and parasitic disease reports, Missouri, five-year median, 1992 and 1993.

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Northeastern, Northwestern, and Southwestern districts. However, the 1993 incidence was still 64.0 percent higher than the five-year median of 411 cases, which was calculated using the annual totals from 1988 to 1992**.** See Figure 2.

Reported *Campylobacter* cases increased from 614 cases in 1992 to 616 cases in 1993. This increase of only 0.3 percent was the smallest since 1987. All districts showed increases in the numbers of reported cases compared to 1992 with the exception of the Northwestern district, which saw a large decrease. The 1993 total of 616 cases was 12.6 percent higher than the five-year median of 547 cases See Figure 2.

Salmonellosis increased in all districts except the Southwestern in 1993. The number of reported cases rose 24.2 percent, from 426 to 529. However, the 1993 total is still 21.7 percent below the five-year median of 676 cases, and reflects a downward trend for Missouri *Salmonella* cases. See Figure 2. The most common serotypes of *Salmonella* reported in 1992 and 1993 are shown in Table 1.

The number of reported cases of *Yersinia enterocolitica* decreased 29.7 percent from 37 cases in 1992 to 26 cases in 1993. These 26 cases are 27.8 percent below the five-year median of 36 cases. See Figure 2. As in previous years, the largest numbers of cases were reported among black children in the Eastern and Northwestern districts.

Parasites

Reported giardiasis cases increased by 4.2 percent, from 739 cases in 1992 to 770 cases in 1993. The 770 cases for 1993 were slightly (2.5%) below the five-year median of 790 cases. See Figure 2. Cases increased during 1993 in the Eastern and Southeastern districts, with decreases being seen in the other areas of the state.

Table 1. Most common *Salmonella* serotypes, Missouri, 1992 and 1993

	1992			1993		
	Serotype	No. of Cases	Percent	Serotype	No. of Cases	Percent
1.	*S. typhimurium*	113	26.5%	*S. typhimurium*	148	28.0%
2.	*S. enteritidis*	47	11.0%	*S. enteritidis*	57	10.8%
3.	*S. heidelberg*	36	8.5%	*S. heidelberg*	37	7.0%
4.	*S. hadar*	20	4.7%	*S. newport*	27	5.1%
5.	*S. braenderup*	11	2.6%	*S. braenderup*	18	3.4%
6.	*S. newport*	11	2.6%	*S. hadar*	13	2.5%
7.	*S. thompson*	10	2.3%	*S. thompson*	11	2.0%
8.	*S. berta*	6	1.4%	*S. montevideo*	10	1.9%
9.	*S. montevideo*	5	1.2%	*S. muenchen*	8	1.5%
	All Others	167	39.2%	All Others	200	37.8%
	Total	**426**		**Total**	**529**	

Figure 3. Hepatitis A cases by month of onset, Eastern District and Missouri, June 1992 thru December 1993.

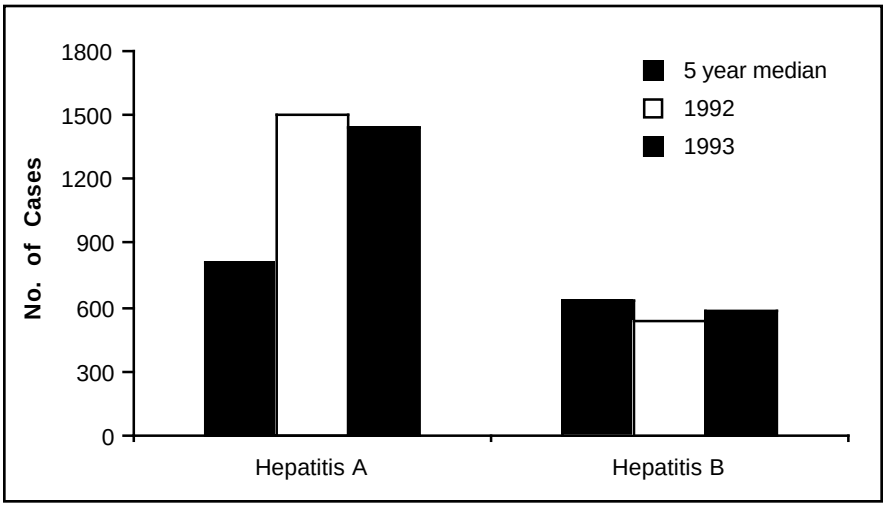

Figure 4. Hepatitis case reports, Missouri, five-year median, 1992 and 1993.

Viral Hepatitis

The number of new cases of hepatitis A reported in the latter half of 1993 dropped dramatically in the Eastern district. See Figure 3. Because of this, the total number of hepatitis A cases for the state was down 3.8 percent from 1,500 in 1992 to 1,443 in 1993. A community-wide outbreak occurred in the Eastern district in the latter half of 1992 and the first half of 1993. This increased the number of cases in the Eastern district from 922 in 1992 to 1,144 in 1993. Reductions in the numbers of reported cases were seen in 1993 in the Northeastern, Southeastern and Southwestern districts. The 1993 total of 1,443 cases is 78.1 percent higher than the five-year median of 810 cases. See Figure 4.

Hepatitis B cases increased by 9.3 percent, from 535 cases in 1992 to 585 cases in 1993. The 1993 total is down 7.6 percent from the five-year median of 633 cases. See Figure 4. All districts reported decreases in the number of reported cases except the Eastern and Southeastern districts. It should be noted that hepatitis surveillance in the Eastern District, where reported hepatitis B cases increased last year by 32.6 percent, has been significantly enhanced in response to the 1992–93 outbreak of hepatitis A. One result of this improved surveillance is that hepatitis B reporting has been much more complete in this region.

Meningitis

There was a slight (6.3%) increase to 34 cases in 1993 from 32 cases in 1992. Thirty-two cases is also the five-year median. See Figure 5. The Northwestern, Southeastern and Southwestern districts reported increases in the number of cases in 1993. The first months of 1994 are demonstrating an increase in reported meningococcal meningitis cases that was not seen in 1993. See Figure 6.

There was little change during 1993 in the number of reported cases of aseptic meningitis**.** The number of cases for the year totaled 275, up 1.1 percent from the cases reported during 1992, and 11.8 percent higher than the five-year median of 246 cases. See Figure 6. Increases were seen in the Central, Northeastern and Southeastern districts. All other districts reported a decrease in the number of cases reported.

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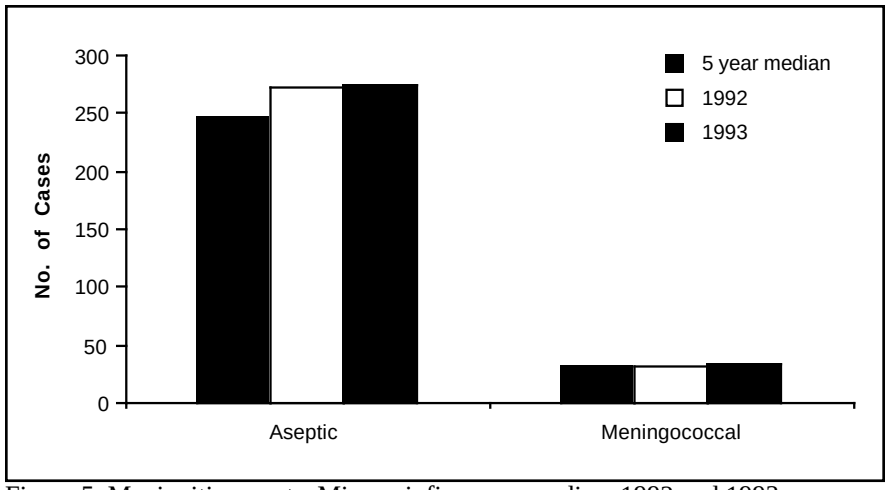

Figure 5. Meningitis reports, Missouri, five-year median, 1992 and 1993.

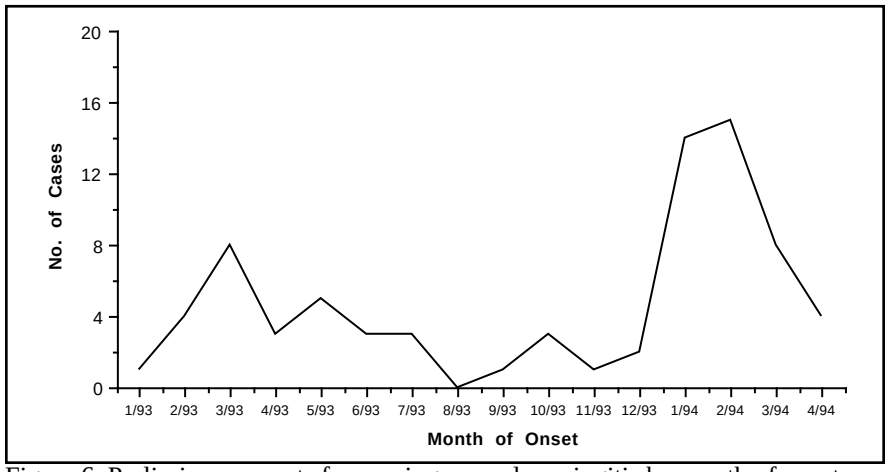

Figure 6. Preliminary reports for meningococcal meningitis by month of onset, Missouri, January 1993 thru April 1994.

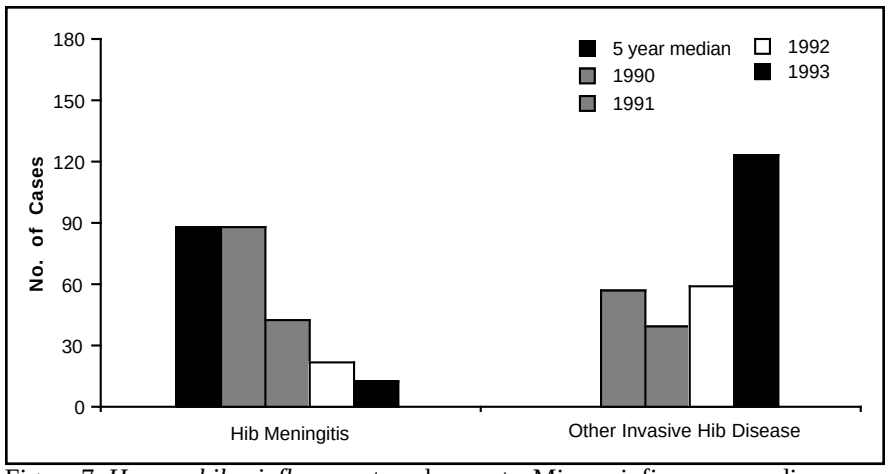

Figure 7. *Haemophilus influenzae* type b reports, Missouri, five-year median, 1990–93.

1993 Outbreaks of Communicable Disease*

Michael Fobbs, B.A.
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Bureau of Communicable Disease Control

In 1993, there were 44 communicable disease outbreaks reported in Missouri involving 1,642 people. This is an increase of 7.3 percent from 41 outbreaks reported in 1992. The number of outbreaks reported annually, 1988–92, ranged from 20 to 49 with a median of 41. These outbreaks involved many different modes of transmission and several widely varying etiologic agents, and they occurred in a variety of settings. The modes of transmission were as follows: 19 were foodborne, 23 were suspected person-to-person transmission, one was waterborne, and in one outbreak the mode of transmission was not determined.

During 1993, communities and restaurants were the most common settings for outbreaks, with each accounting for 12 (27.3%) of the reported outbreaks, schools were second with six outbreaks (13.6%); day care settings were involved in three outbreaks (6.8%); homes, camps and hotels accounted for two outbreaks (4.5%) each. A catered event was responsible for only one outbreak (2.3%), unlike 1992 when there were six such events. One outbreak each occurred in a prison, a grocery store, a women's and children's shelter and a recreational area. The outbreaks are shown in Table 1 by cause, setting and number of cases.

The largest single event was a community-wide outbreak of waterborne *Salmonella typhimurium* which affected approximately 500 individuals. It was described in the March/April 1994 issue of the *Missouri Epidemiologist.*

The largest proportion of outbreaks reported during 1993 consisted of acute gastrointestinal illness of unknown etiology (AGI). Nineteen outbreaks of AGI, affecting 823 people, were reported. Foodborne transmission was the most common mode, being implicated in 14 of these outbreaks. The other five AGI outbreaks resulted from person-to-person transmission. AGI outbreaks occurred in the following settings: ten restaurants, four schools, one hotel, one catered event, one summer camp, one prison and one grocery store deli.

Table 1. Communicable disease outbreaks by cause, setting and number of cases, Missouri, 1993

Disease/ Mode of Transmission	No. of Outbreaks	Setting	No. of Cases
AGI*			
Foodborne	14	CT, H, O, P, 10R	264
Person-to-Person	5	CA, 4S	559
Total	**19**		**823**
Shigellosis			
Person-to-Person	6	3C, 2DC, O	98
Foodborne	2	2FG	25
Unknown	1	O	4
Total	**9**		**127**
Hepatitis A			
Person-to-Person	6	5C, CA	70
Salmonella			
Foodborne	2	H, R	46
Waterborne	1	C	500
Total	**3**		**546**
ARI**	**2**	**C**	**40**
Scabies	**2**	**DC, S**	**9**
Aseptic meningitis	**1**	**C**	**3**
Staphylococcus aureus			
Foodborne	**1**	**R**	**8**
Erythema infectiosum	**1**	**S**	**16**
TOTAL	**44**		**1,642**

*Acute gastrointestinal illness of unknown etiology
**Acute respiratory illness of unknown etiology

Key

C	Community	F	Family Garthering	R	Restaurant
CA	Camp	H	Hotel	S	School
CT	Catered Event	O	Other	W	Workplace
DC	Daycare	P	Prison		

***excludes outbreaks related to HIV, sexually transmitted diseases, tuberculosis, vaccine-preventable diseases and zoonotic diseases. These disease outbreaks are covered in other articles contained in this issue.**

Shigellosis was reported as the causative agent for nine outbreaks involving 127 people. Two outbreaks were transmitted by food, and one involved exposure to a recreational area. The other six outbreaks of shigellosis involved person-to-person transmission. Settings for shigellosis outbreaks included three communities, two family gatherings, two day care centers, one recreational area and one women's and children's shelter.

Six hepatitis A outbreaks affecting 70 people were reported. In each instance, transmission of the virus was from person to person. The settings were five communities and one day camp.

Salmonellosis was the causative agent in three outbreaks that affected an estimated 546 people. There were two foodborne outbreaks, one in a hotel and one in a restaurant. The third outbreak caused by *salmonella* was the community-wide waterborne outbreak referred to above.

Two outbreaks of acute respiratory illness of unknown etiology (ARI) were reported, affecting a total of 40 people. Each occurred in a community setting.

Scabies was the causative agent in two outbreaks involving a total of nine people in a school and a daycare center. *Staphylococcus aureus* food poisoning affected eight people who ate in a restaurant. An outbreak of erythema infectiosum involved 16 people in a school, and a cluster of three aseptic meningitis cases occurred in a small community.

Table 2. Nosocomial outbreaks and investigations by cause, setting and number of cases, Missouri, 1993

Disease/ Mode of Transmission	No. of Outbreaks	Setting	No. of Cases
Scabies	19	H,18NH	215
AGI*			
Person-to-Person	10	10NH	536
Influenza	4	4NH	166
Staphylococcus aureus			
Person-to-Person	2	H**, NH	16
Environmental	1	H**	6
Total	**3**		**22**
Clostridium difficile	3	H, 2NH	25
ARI***	2	2NH	69
Pediculosis	2	2NH	48
Citrobacter			
Medical Procedure	1	O	5
Conjunctivitis	1	NH	9
Influenza-like	1	H	40
TOTAL	**46**		**1,135**

* Acute gastrointestinal illness of unknown etiology
** Methicillin resistant *Staphylococcus aureus*
*** Acute respiratory illness of unknown etiology

Key
H Hospital
NH Nursing Home
O Other Health Care Facility

1993 Nosocomial Outbreaks

Hospitals, nursing homes and other long-term-care facilities in Missouri reported 46 institutionally-acquired (nosocomial) outbreaks of communicable disease during 1993. Altogether, 1,135 cases of illness were reported. The number of nosocomial outbreaks reported annually ranged from 11 to 49 during the period 1988–92, with a median of 37.

In 44 of the 46 (95.6%) outbreaks, transmission of disease was from person to person, in one outbreak, transmission was suspected to have occurred through environmental exposure, and in one outbreak, transmission occurred via a medical procedure. Nursing homes were the setting for 40 (87.2%) of the outbreaks, hospitals for five (10.8%), and one outbreak (2.1%) was in another health care setting. Table 2 describes the outbreaks by cause, setting and number of cases.

Scabies accounted for 19 (41.3%) of the 46 outbreaks, and involved 215 people. Eighteen of these scabies outbreaks occurred in nursing homes, and one occurred in a hospital. In all instances, transmission of the mite was from person to person.

Outbreaks of acute gastrointestinal illness of unknown etiology (AGI) occurred in ten nursing homes and affected a total of 536 persons. The mode of transmission in each instance was person to person.

Four confirmed influenza outbreaks involving 166 people were reported in nursing homes. One outbreak of influenza-like illness (40 cases) was reported in a hospital.

Three outbreaks of staphylococcal infection were reported, affecting a total of 22 persons. Two of these outbreaks occurred in hospitals, the other took place in a nursing home. The two hospital outbreaks involved methicillin-resistant *Staphylococcus aureus*. One of these outbreaks consisted of a cluster of

(continued on page 11)

Bureau of Environmental Epidemiology FY93 Report

Gale M. Carlson, B.S.
Bureau of Environmental Epidemiology

The Bureau of Environmental Epidemiology is routinely involved in assessing risk to human health from hazardous substances in the environment. Requests come from private citizens, district and local health authorities, physicians, various municipal agencies, other state agencies and various federal organizations. A variety of documents discussing exposure levels, health effects, safe clean-up levels and risk from exposure to substances at hazardous waste sites throughout Missouri are produced for the Missouri Department of Natural Resources, the U.S. Environmental Protection Agency (EPA) and the Agency for Toxic Substances and Disease Registry (ATSDR).

In 1993, all 51 abandoned and uncontrolled hazardous waste facilities in the state were reassessed for their risks to human health. Another 21 assessments were conducted on candidate hazardous waste facilities. See Figure 1. The Health Assessment Program in cooperation with ATSDR has initiated or completed five health assessments. The risk assessment program in cooperation with EPA completed five risk assessments and numerous risk assessment consultations. Based on the success of our Risk Assessment Program, EPA has begun funding two other states to work cooperatively with them to assess risks at Superfund sites.

In cooperation with the Bureau of Health Data Analysis, four Resource Conservation and Recovery Act Health Profiles were reviewed. These are profiles of the health status of a community surrounding a proposed resource recovery facility such as battery recycling, electrical equipment refurbishing or waste incineration. Clean-up assessments (development of safe residual contaminant levels) for 18 sites in the state were also produced.

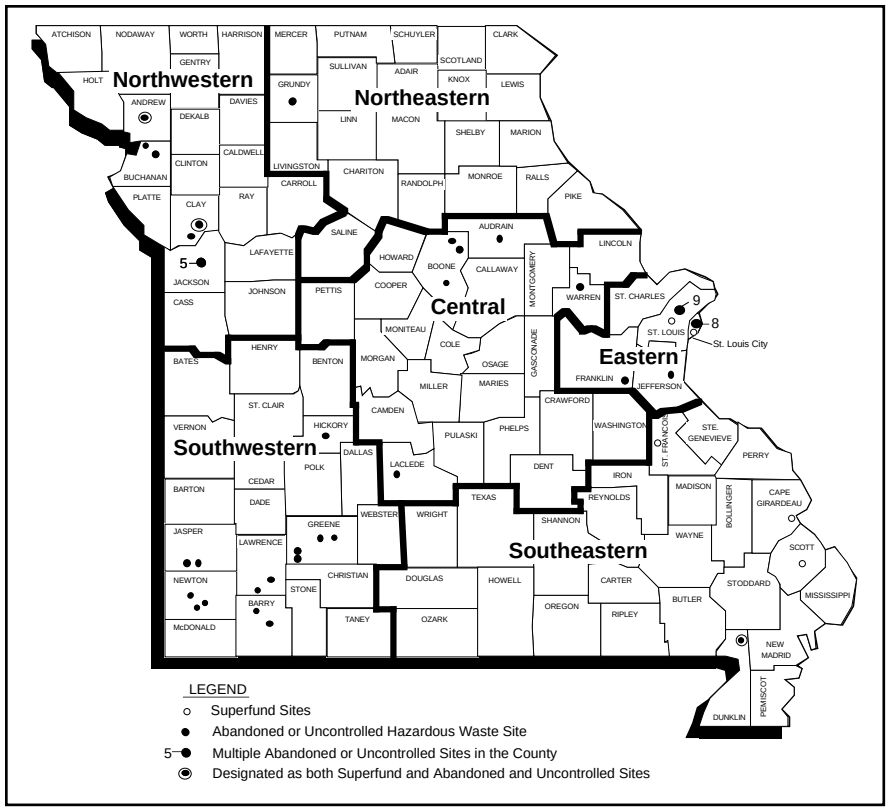

Figure 1. Abandoned or uncontrolled hazardous waste sites and Superfund sites addressed by the Bureau of Environmental Epidemiology during 1993, Missouri.

Our Industrial Hygiene Program was terminated in the spring of 1993 because of legislatively mandated budgetary cutbacks. Between May 1993, when that program ended, and December 31, 1993, the bureau received more than 150 requests for help on indoor air quality problems. The bureau has been trying to aid those persons by referring them to private consultants, but many of those persons' needs were not met.

The Missouri Occupational Fatality Assessment and Control Evaluation Program (MO FACE) develops detailed epidemiological investigation protocols for fatalities resulting from falls, electrocutions, farming and asphyxiation deaths caused by entry into confined spaces. During 1993, MO FACE conducted 18 fatality investigations and worked with the companies involved in these fatalities to prevent similar incidents. Occupational fatality reports were produced for each of these investigations and disseminated to safety groups in the United States and Missouri. The program was notified of 107 traumatic work-place fatalities during federal fiscal year 1993 (FFY93), October 1, 1992 through September 30, 1993. See Figures 2 and 3. A MO FACE annual report summarizing all the activities of the program to date was disseminated.

The Lead Program coordinates all Department of Health lead-related programs, which includes the Centers for Disease Control and Prevention (CDC) Childhood Lead Poisoning Prevention and Control Program, EPA Region VII Lead Training and Outreach Grant Programs, the Medicaid Lead Screening Program and the State Lead In-Service Training for Local Health Departments.

In June 1993, Senate Bill 232 was signed into law (Chapters 701.300–338, RSMo) establishing the Commission on Lead Poisoning, which consists of 21 members appointed by the Governor of the State of Missouri. The Commission shall submit a report to the General Assembly and the Governor with recommendations on how to eliminate childhood lead poisoning by the year 2012. The bureau provided technical and logistical support for this commission.

In addition, the new statutes require the reporting of elevated blood lead levels by providers and laboratories; accreditation and certification programs for lead abatement contractors to be established along with state regulations; and the establishment and maintenance of a lead poisoning information reporting system that will track Missouri lead poisoning cases and the appropriateness of lead abatement programs.

In July 1993, the Childhood Lead Poisoning Prevention and Control Program was awarded a grant from CDC. Funding from this grant is aimed at the lead programs in St. Louis City and St. Louis County to purchase needed equipment and to hire additional staff members.

The bureau also received an EPA Region VII Training Grant with St. Louis University School of Public Health being the subcontractee. This grant establishes lead training (inspector) and educational outreach activities for the St. Louis metropolitan area.

As of June 1, 1994, there have been 30,193 reported blood lead tests on children less than six years of age. The breakdown includes St. Louis City with 17,850 screens, St. Louis County with 4,506 screens, Springfield with 2,606 screens, Kansas City with 2,877 screens and outstate-Missouri (local health departments) with 2,354 screens.

The Missouri Hazardous Substances Emergency Events Surveillance (HSEES) system monitors hazardous substances emergency events to allow a better understanding of the public health impact of such events so that the morbidity from these events in Missouri can be reduced. The funding for HSEES was received in from ATSDR in October 1993. In the remainder of 1993, efforts were directed toward identifying sources of information about hazardous substances releases. The main agency of contact is the Environmental Services Program within the Missouri Department of Natural Resources. Other sources of information include the Division of Fire Safety, the Missouri Highway Patrol and the State Emergency Management Agency. During this period, 129 events were investigated; however, only 38 met the HSEES case definition.

The bureau issues an annual Fish Consumption Advisory. The May 1993 advisory emphasized that carp and catfish in many water bodies in the state were still contaminated with chlordane and other pesticides at a level of health concern. However, the advisory was simplified and shortened because of results obtained in a 1991 study by the bureau. The study revealed that previous advisories, based on levels of contaminants in fish did not accurately predict exposure of humans to the contaminants. The bureau discovered that the factors causing elevated blood contamination were not influenced by where the fish were caught but by the amount of fish consumed and the length of time the fish were consumed at those levels. The advisory also emphasized the types of fish that were safe to eat and stressed the benefits of eating fish as a good healthy protein source.

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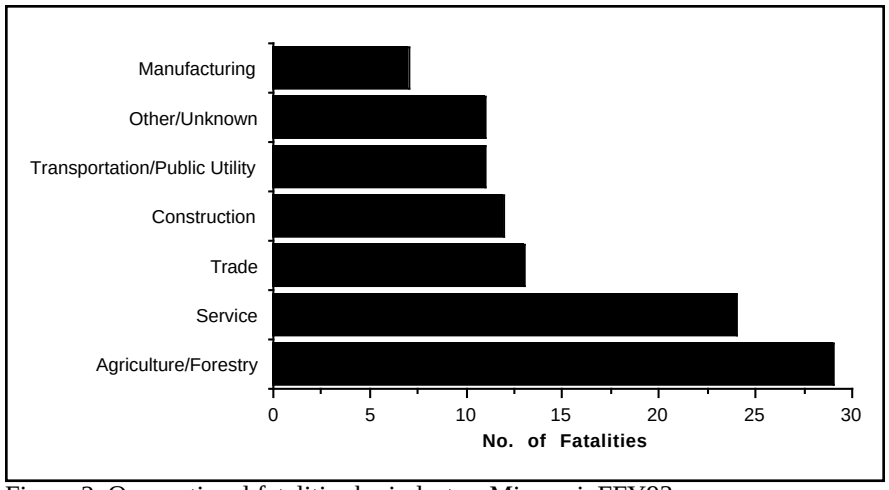

Figure 2. Occupational fatalities by industry, Missouri, FFY93.

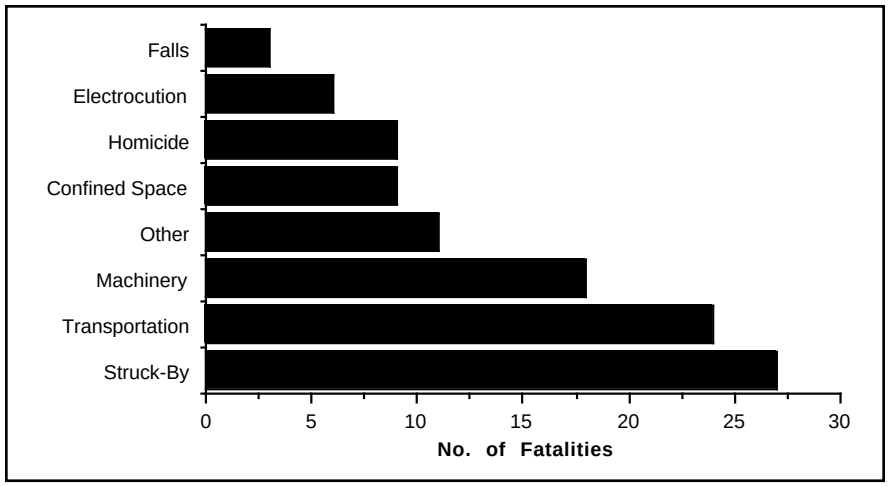

Figure 3. Causes of occupational fatalities, Missouri, FFY 93.

1993 HIV/AIDS Annual Report

Wendy Watkins
Bureau of STD/HIV Prevention

On January 1, 1993, the Centers for Disease Control and Prevention expanded the AIDS case definition to include persons who are infected with HIV and have laboratory evidence of severely impaired immune function. The expanded definition emphasizes the clinical importance of the CD4+ lymphocyte count in monitoring HIV disease by including under the definition of AIDS all HIV-infected persons with CD4+ cell counts under 200 and/or a CD4+ percent of <14. Also, the new definition adds pulmonary tuberculosis, recurrent pneumonia and invasive cervical cancer to the previous list of 23 AIDS-indicator diseases or conditions.

In addition to the changes in the number of reported AIDS cases associated with the expansion of the AIDS case definition, there has also been a change in the way in which Missouri HIV data is reported. Persons meeting the AIDS surveillance definition are no longer included in the HIV data. This makes it possible to more accurately depict the epidemic and better describe the HIV disease continuum for Missouri.

The number of cases of AIDS reported in Missouri has increased each year since it became a reportable disease in 1983. See Figure 1. The expansion of the surveillance case definition had a significant impact on the number of AIDS cases reported for Missouri in calendar year 1993. Of the 1,664 AIDS cases reported during this period, 1,024 (61.5%) were the direct result of the new case definition. There were 677 cases of HIV infection reported in 1993.

In 1993, the largest number of AIDS and HIV infections occurred in the 20–29 and 30–39 year age groups. For HIV, 82.3 percent (557) of the reported infections occurred in the 20–29 and 30–39 year age groups. For AIDS, 70.7 percent (1,177) of the reported cases occurred in the same age groups. See Figure 2.

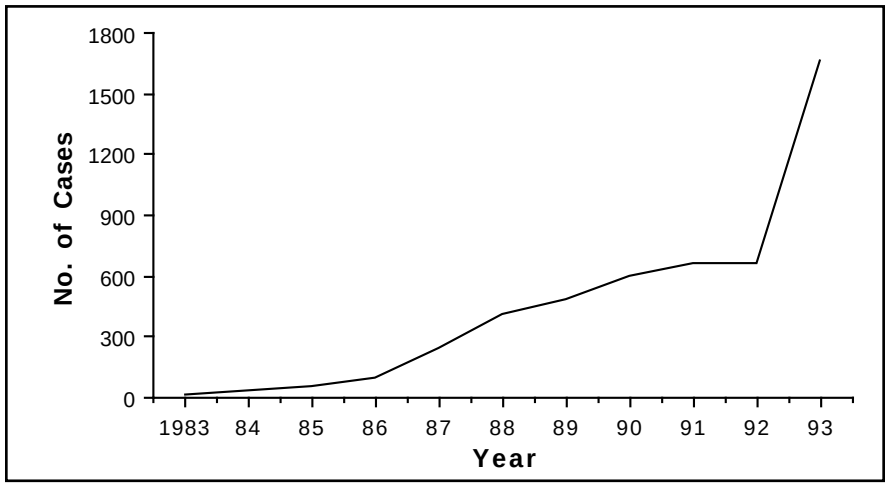

Figure 1. AIDS cases by year, Missouri, 1983–93

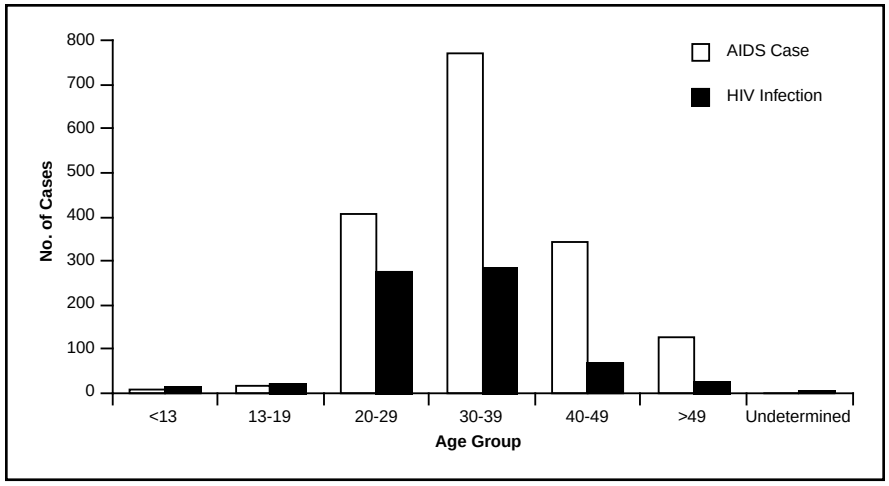

Figure 2. AIDS cases and HIV infection incidence by age group, Missouri, 1993

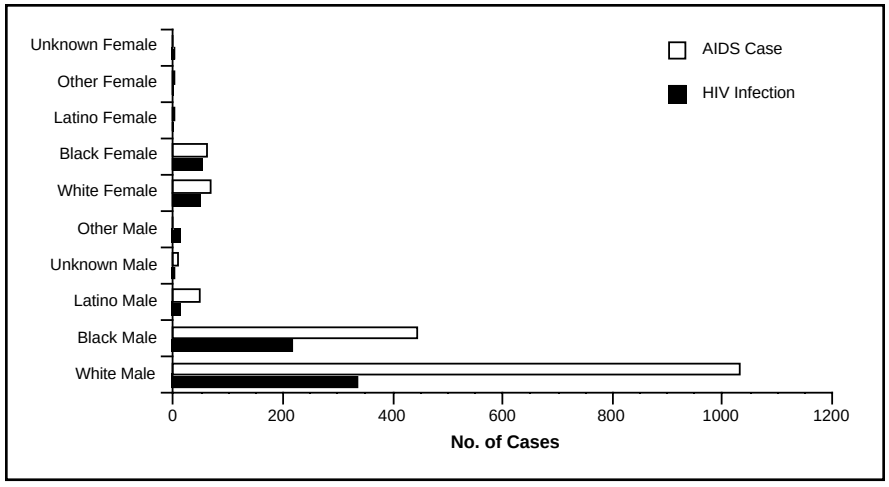

Figure 3. AIDS cases and HIV infection incidence by race, Missouri, 1993

White males comprised the majority of reported HIV/AIDS cases, 49.6 percent (336) for HIV, and 62.0 percent (1,031) for AIDS cases. Black males formed the second largest group, with 31.8 percent (215) reporting HIV infection and 26.6 percent (443) reporting AIDS. See Figure 3.

The most often reported risk factor for 1993 was homosexual/bisexual activity with 68.2 percent (1,135) of the AIDS cases being associated with this exposure category; 56.3 percent (381) of HIV infections in 1993 were in the homosexual/bisexual risk category. Heterosexual contact was the second largest risk factor for HIV in 1993 at 13.7 percent (93). For AIDS, 7.0 percent (114) of the cases in 1993 reported heterosexual contact as the risk factor. See Figure 4.

The predominate areas for reported cases of AIDS in 1993 were the major metropolitan areas of St. Louis and Kansas City. See Figure 5. These combined areas reported 76.3 percent (1,270) of the total AIDS cases for the state.

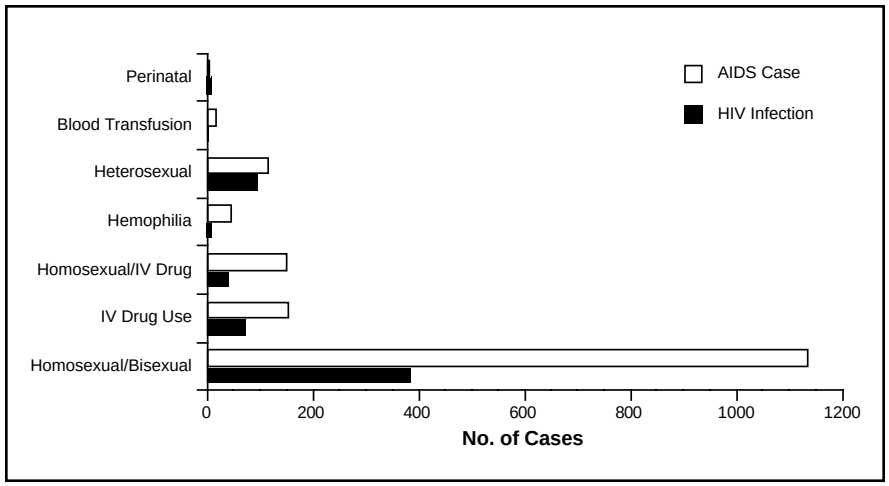

Figure 4. AIDS cases and HIV infection incidence by sexual activity, Missouri, 1993

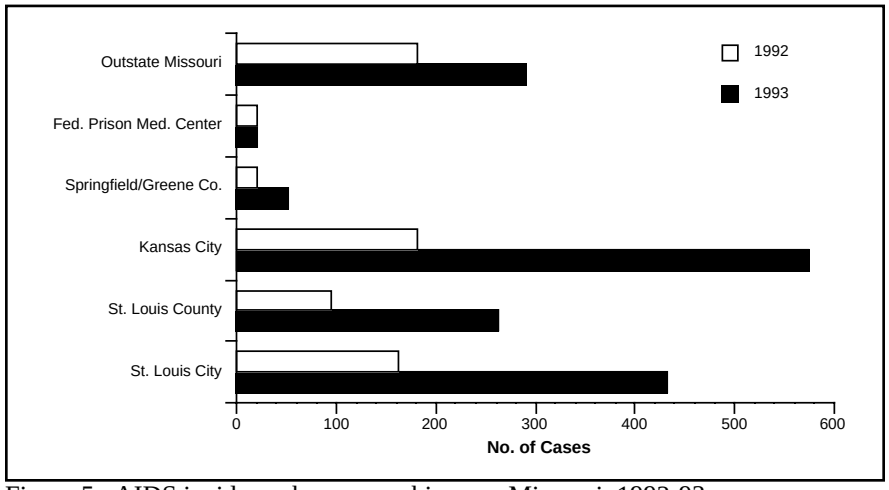

Figure 5. AIDS incidence by geographic area, Missouri, 1992-93

HIV Telephone Consultation Service - (800) 933-3413

Recognizing the importance of providing clinicians with quick and relevant clinical HIV education and case consultation, the Health Resources and Services Administration's (HRSA) Community Provider AIDS Training (CPAT) Project at San Francisco General Hospital offers a telephone consultation service to health care providers. By supporting primary health care providers in caring for their patients with asymptomatic and symptomatic HIV disease, more patients will be able to remain in their communities with their own medical providers and social support networks.

The service is called a "warmline" because it is not staffed 24 hours a day. Voice mail is available 24 hours a day, seven days a week.

When You Call This Service:
- The phone is answered by either a clinical pharmacist, physician or nurse practitioner.
- Whenever possible, your question is answered immediately. Most calls are answered within two hours. Almost all calls are answered within a day.
- Patient-specific information (CD-4 cell count, current medications, previous manifestations of HIV disease, sex, age and transmission category) is requested for case consultation.
- Written materials supporting the telephone discussion are sent by mail or facsimile whenever needed.

Hours for the Warmline
7:30 a.m. - 5:00 p.m. PST
Monday - Friday

Who Should Call:
- Physicians
- Nurse Practitioners
- Pharmacists
- Dentists
- Other health care workers involved in primary care of HIV-infected persons

The Warmline Provides:
- case consultation
- drug information regarding HIV/AIDS medications
- infection control and HIV prevention information
- clinical trials information
- subspecialty care referral information
- literature searches

This service is provided free of charge.

Sexually Transmitted Diseases - 1993

Bill Huber
Bureau of STD/HIV Prevention

The Bureau of STD/HIV Prevention provides assistance to local health departments for the control of sexually transmitted diseases in their communities. Guidelines for testing, diagnosis and treatment are developed and distributed as recommended by the Centers for Disease Control and Prevention. Screening materials and medications for treatment are provided to local health departments and bureau personnel are available to assist with partner notification and follow-up services. The bureau also provides morbidity-trend analysis and other program evaluation services.

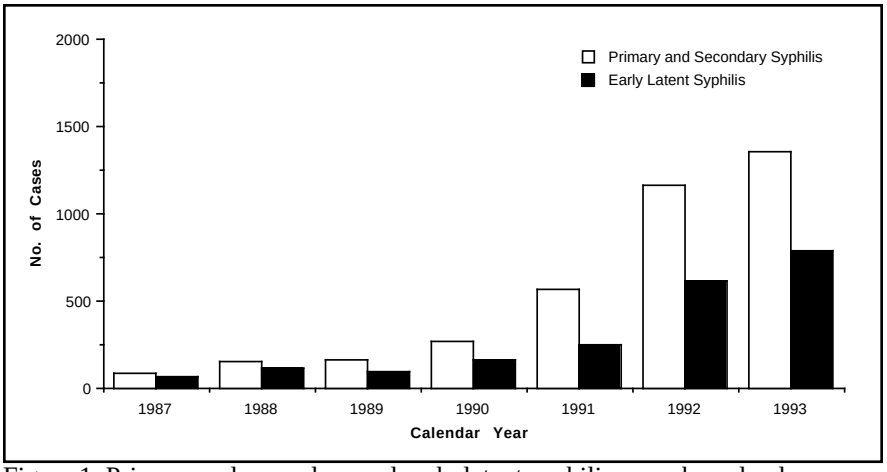

Figure 1. Primary and secondary and early latent syphilis cases by calendar year, Missouri, 1987–93.

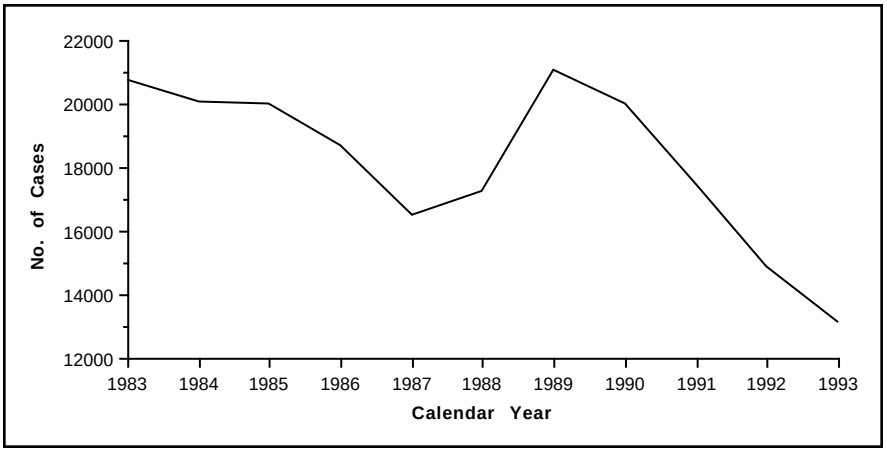

Figure 2. Gonorrhea cases by calendar year, Missouri, 1983–93.

Early Syphilis

Primary, Secondary and Early Latent (of less than one year's duration)

The reported incidence of early syphilis increased significantly in Calendar Year (CY) 1992 compared to CY 1991. Increases were also noted in CY 1993. See Figure 1. Primary and secondary cases increased by 16 percent from 1,167 in 1992 to 1,354 in 1993. Early latent cases increased by 27 percent from 620 in 1992 to 790 in 1993. St. Louis City reported almost half of the early syphilis cases in Missouri; 908 primary and secondary cases and 423 early latent cases. St. Louis County also reported an increase in the number of cases identified during CY 1993; with 222 cases of primary and secondary reported along with 135 cases of early latent.

While the increases noted in CY 1992 and 1993 are each significant, primary and secondary syphilis has increased from a 30 year low of 90 cases in CY 1987 to 1,354 (1,404 %) in CY 1993.

A large percentage of syphilis cases in all areas of the state continue to appear related to crack-cocaine use. Usage of this drug also appears to be related to increases in other sexually transmitted diseases.

The primary and secondary syphilis rate of 25.8 per 100,000 population in Missouri during 1993 is significantly higher than the corresponding national rate of 10.6 per 100,000 population.

The primary and secondary syphilis rate in St. Louis City during CY 1992 was 153.3 per 100,000 population. This rate ranks St. Louis number one among cities over 200,000 populations in CY 1992. The rate of 64.8 ranks Kansas City number six in this same category during this same time period.

Gonorrhea

The reported incidence of gonorrhea in Missouri decreased by 11.7 percent from 14,887 cases in CY 1992 to 13,147 in CY 1993. The rate decreased from 286.7 per 100,000 in 1992 to 251.2 per 100,000 in 1993. See Figure 2. St. Louis City reported a decrease in gonorrhea incidence of 6.4 percent. St. Louis County, Kansas City and Outstate Missouri also reported decreases of 13.0, 23.5 and .05 percent respectively. This is the fourth consecutive year in which such decreases in gonorrhea have been reported. This trend appears to be accurate and is supported by a decrease in the positivity

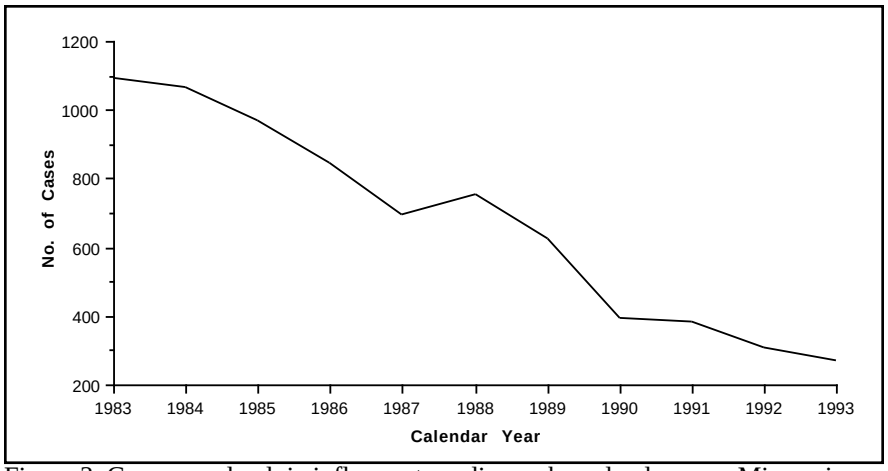

Figure 3. Gonococcal pelvic inflammatory disease by calendar year, Missouri, 1983–93.

observed in the gonorrhea screening project even though a reduction in the level of screening has resulted in a concentration of screening efforts in the highest risk populations.

Penicillinase-producing *N. gonorrhoeae* (PPNG)

All gonorrhea in Missouri is considered to be resistant to penicillin and tetracycline-based medications. Ceftriaxone or Ciprofloxacin are the drug regimens of choice at this time.

Gonococcal Pelvic Inflammatory Disease (GPID)

GPID cases decreased from 308 reported in CY 1992 to 269 reported in CY 1993. This decrease occurred in all areas of the state. St. Louis City reported a decrease of 14 cases, St. Louis County 2, Kansas City 28 and outstate Missouri 11 cases. See Figure 3.

Non-Gonococcal Urethritis (NGU)

Reported cases of NGU decreased six percent from 6,874 in 1992 to 6,425 in 1993. This decrease was noted in all areas of the state.

Chlamydia trachomatis Infections

Reported *Chlamydia trachomatis* infections decreased two percent from 11,907 cases in CY 1992 to 11,625 cases in CY 1993. Widespread therapy of symptomatic patients without testing plus dual treatment of all gonorrhea cases (gonorrhea therapy plus chlamydia therapy), have contributed to under-reported morbidity. Positivity in the screening program has decreased from 16 percent positivity five years ago to ten percent last year.

Genital Herpes

Genital herpes increased very little, with 3,681 cases reported in CY 1992 and 3,729 cases reported in CY 1993.

Congenital Syphilis

Congenital syphilis increased from 28 cases reported in CY 1992 to 97 cases reported in CY 1993. This trend is expected to continue over the next few years due to the increases of early syphilis reported among females, and also to the revised and expanded surveillance criteria for congenital syphilis. The expanded criteria, which went into effect July 1, 1990, lead to some infants with presumptive evidence of congenital syphilis being counted as cases.

St. Louis reported 66 (68%) of the congenital syphilis cases in CY 1993. St. Louis County and Kansas City reported 12 (12%) and 11 (11%) respectively.

1993 Nosocomial Outbreaks

(continued from page 5)
pneumonias, the other involved multiple body sites. The nursing home outbreak also involved multiple body sites.

Three outbreaks of *Clostridium difficile* diarrhea, involving a total of 25 people, occurred in two nursing homes and a hospital. Two outbreaks of acute respiratory illness of unknown etiology (ARI), affecting a total of 69 people, occurred in nursing homes.

Two outbreaks of pediculosis (48 cases) and one outbreak of conjunctivitis (9 cases) also occurred in nursing homes.

An outbreak of five cases of *Citrobacter* bacteremia was associated with renal dialysis procedures in one facility.

A nursing home was involved in the community-wide waterborne *S. typhimurium* outbreak mentioned in the communicable disease section.

Bureau of Environmental Epidemiology FY93 Report

(continued from page 7)
Results of a cooperative study between the bureau and ATSDR to determine if there were statistically significant differences between the respiratory functions of two groups of persons around lead smelters in Missouri was to be completed in the summer of 1993. Although all data collection was completed as planned by fall of 1992, ATSDR has yet to finish the analysis and write up on this study. The most recent information indicates that those results may be released in 1994.

In cooperation with ATSDR, EPA and St. Louis University, the bureau began an investigation in 1991 of persons exposed to lead and zinc mining wastes in the Jasper County Superfund Site area. The report of this study was released by the bureau in May 1994 and will be reprinted in the next issue of this newsletter. Copies of the report can be obtained by contacting the bureau.

1993–94 Influenza Summary

Irene Donelon
Bureau of Communicable Disease Control

There were a total of 283 laboratory-confirmed cases of influenza reported in Missouri during the 1993–94 season. All were type A with 53 subtyped as A/Beijing (H3N2). No cases of type B influenza were reported in Missouri this season.

The 1993–94 influenza season was characterized by outbreaks in a variety of settings. Outbreaks of influenza-like illness were reported in ten schools with seven closings; the outbreak in a Jackson County school was laboratory confirmed as A/Beijing. A community-wide outbreak of influenza-like illness was reported in Putnam County and confirmed as A/Beijing. Unconfirmed outbreaks were reported in a correctional facility and a state hospital. Eight outbreaks were reported in long-term care facilities with three confirmed as A/Beijing and one as type A (not subtyped). Attack rates in long-term care facilities ranged from 20–57 percent. Many of the cases occurred in immunized individuals.

The first culture-confirmed case of influenza (type A/Beijing) was reported on November 23, 1993 in a four year old child in St. Louis County. Influenza-like illness peaked during week two of 1994. See Figure 1. This was three weeks earlier than the previous seven-year average and in line with predictions by the Centers for Disease Control and Prevention (CDC). Laboratory confirmation of influenza cases peaked in week four of 1994. See Figure 2.

Pneumonia and influenza deaths were above the previous ten year average for most of the season and peaked during week four of 1994. See Figure 3. This reflected the nationwide trend. High mortality rates are not uncommon in seasons when type A (H3N2) influenza is circulating. The Division of Health

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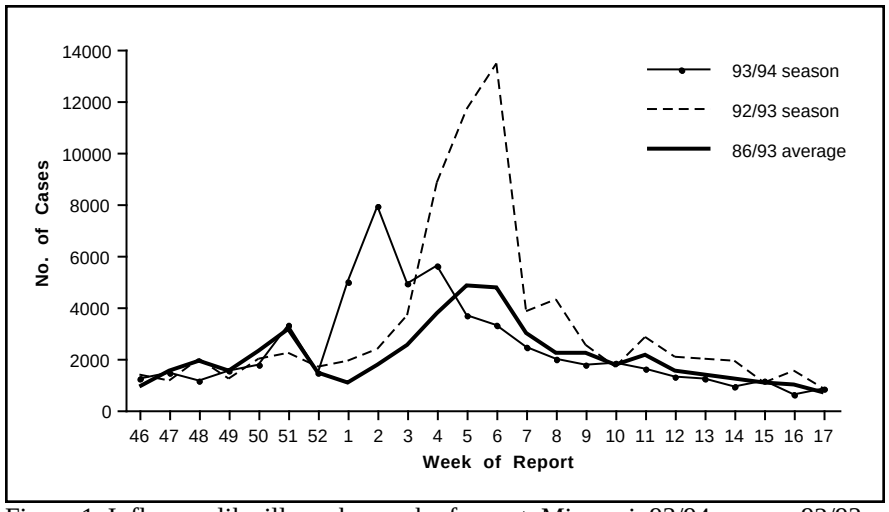


Figure 1. Influenza-like illness by week of report, Missouri, 93/94 season, 92/93 season and 86/93 average.

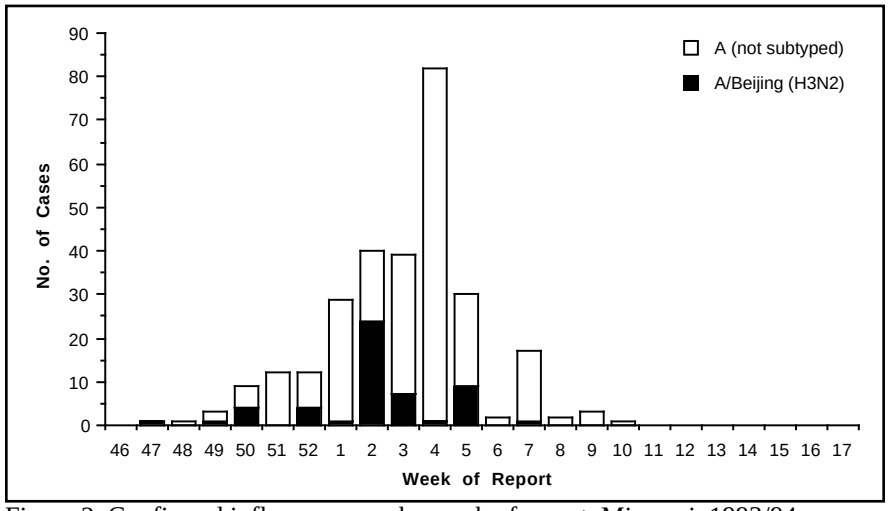


Figure 2. Confirmed influenza cases by week of report, Missouri, 1993/94 season.

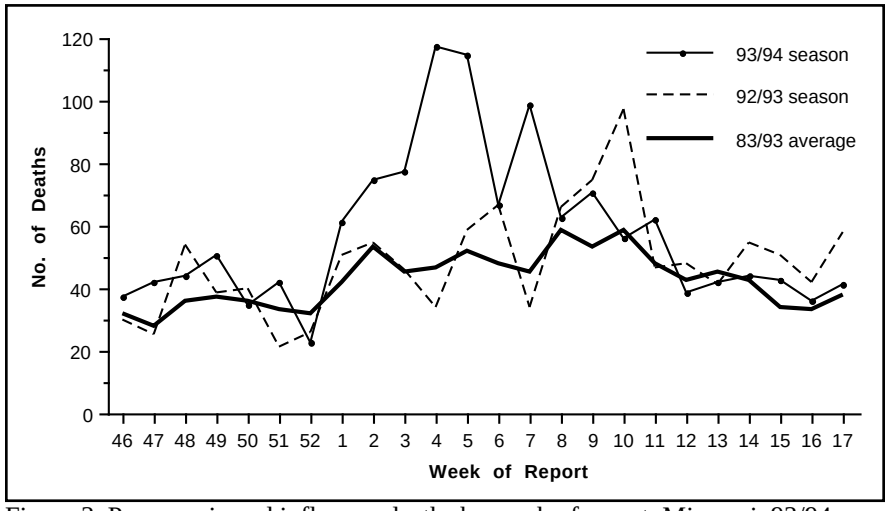
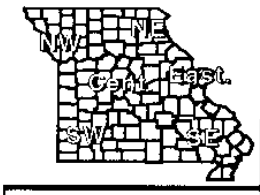


Figure 3. Pneumonia and influenza deaths by week of report, Missouri, 93/94 season, 92/93 season and 83/93 average.



Missouri Department of Health
Disease Prevention - Communicable Disease Control
BIMONTHLY MORBIDITY REPORT

Reporting Period *
March-April, 1994

	Districts							KANSAS CITY	ST. LOUIS CITY	ST. LOUIS CO.	SPGFLD GREENE CO.	2 MONTH STATE TOTALS		CUMULATIVE		
	** NW	NE	CD	SE	** SW	** ED	*** OTHER					1994	1993	FOR 1994	FOR 1993	5 YR MEDIAN
Vaccine Preventable Dis.																
Chickenpox	928	220	276	898	401	552		0	1	16	11	3303	3159	5728	5414	5414
Diphtheria	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Hib Meningitis	0	0	0	0	0	0		2	0	0	0	2	1	5	4	18
Hib Other Invasive	1	0	1	0	2	1		5	1	2	1	14	18	22	35	##
Influenza	1	0	1	0	2	0		3	2	1	0	10	87	156	228	178
Measles	0	0	0	0	0	0		0	0	0	0	0	1	0	1	1
Mumps	3	1	1	0	0	0		1	0	0	0	6	7	10	12	17
Pertussis	0	2	1	0	1	0		0	1	0	0	5	6	11	16	14
Polio	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Rubella	0	0	0	0	0	0		0	0	0	0	0	0	0	1	1
Tetanus	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Viral Hepatitis																
A	5	1	2	0	1	3		7	31	14	8	72	290	153	679	203
B	5	2	6	6	3	0		10	32	8	3	75	114	139	205	192
Non A - Non B	0	0	0	0	0	0		0	0	1	1	2	9	4	14	12
Unspecified	0	0	0	0	0	0		0	0	0	0	0	5	0	8	5
Meningitis																
Aseptic	3	0	2	3	1	2		2	0	3	3	19	13	38	31	28
Meningococcal	5	0	1	1	1	3		2	1	0	0	14	10	39	14	14
Other	3	0	1	4	0	3		0	1	2	1	15	8	24	25	20
Enteric Infections																
Campylobacter	9	4	6	9	2	9		3	10	25	2	79	60	118	114	114
Salmonella	8	0	10	8	6	7		7	3	10	2	61	65	112	101	108
Shigella	0	0	49	1	1	5		6	3	9	5	80	103	109	191	133
Typhoid Fever	0	0	0	0	0	0		0	0	0	0	0	1	0	1	1
Parasitic Infections																
Amebiasis	1	0	0	2	0	0		0	1	0	0	4	4	8	10	7
Giardiasis	6	4	16	16	4	5		14	2	9	2	78	85	150	172	172
Sexually Transmitted Dis.																
AIDS	12	1	16	6	5	3	6	37	33	19	4	142	405	263	1175	173
Gonorrhea	81	13	76	83	40	18		533	1015	386		2245	1655	3694	3466	5282
Genital Herpes	38	21	46	38	67	50		148	127	117		652	639	1159	1244	1125
Nongonoc. urethritis	24	13	10	27	7	1		303	677	33	8	1103	1045	2038	1940	2139
Prim. & Sec. syphilis	0	0	2	3	1	1		16	123	43		189	225	368	445	148
Tuberculosis																
Extrapulmonary	0	1	0	2	2	0	0	2	1	0	1	9	3	11	6	10
Pulmonary	0	1	4	10	3	0	0	5	3	8	1	37	41	57	59	59
Zoonotic																
Animal Bites	168	38	73	168	145	178		0	0	368	24	1162	1074	1812	1736	1512
Psittacosis	0	0	0	0	1	0		0	0	0	0	1	0	1	0	0
Rabies (Animal)	0	0	0	3	1	0		0	0	0	1	4	0	6	1	6
Rocky Mtn. Sp. Fever	0	0	0	0	0	0		0	0	0	0	0	2	0	2	1
Tularemia	0	0	0	0	1	0		0	0	0	1	1	2	2	3	3

Low Frequency Diseases

Anthrax
Botulism
Brucellosis
Chancroid
Cholera
Cryptosporidiosis
Encephalitis (infectious) - 2
Encephalitis (viral/arbo-viral)
Granuloma Inguinale
Kawasaki Disease - 2
Legionellosis - 13
Leptospirosis
Lymphogranuloma Venereum - 1
Malaria - 3
Plague
Rabies (human)
Reye's Syndrome
Rheumatic fever, acute - 1
Toxic Shock Syndrome - 3
Trichinosis

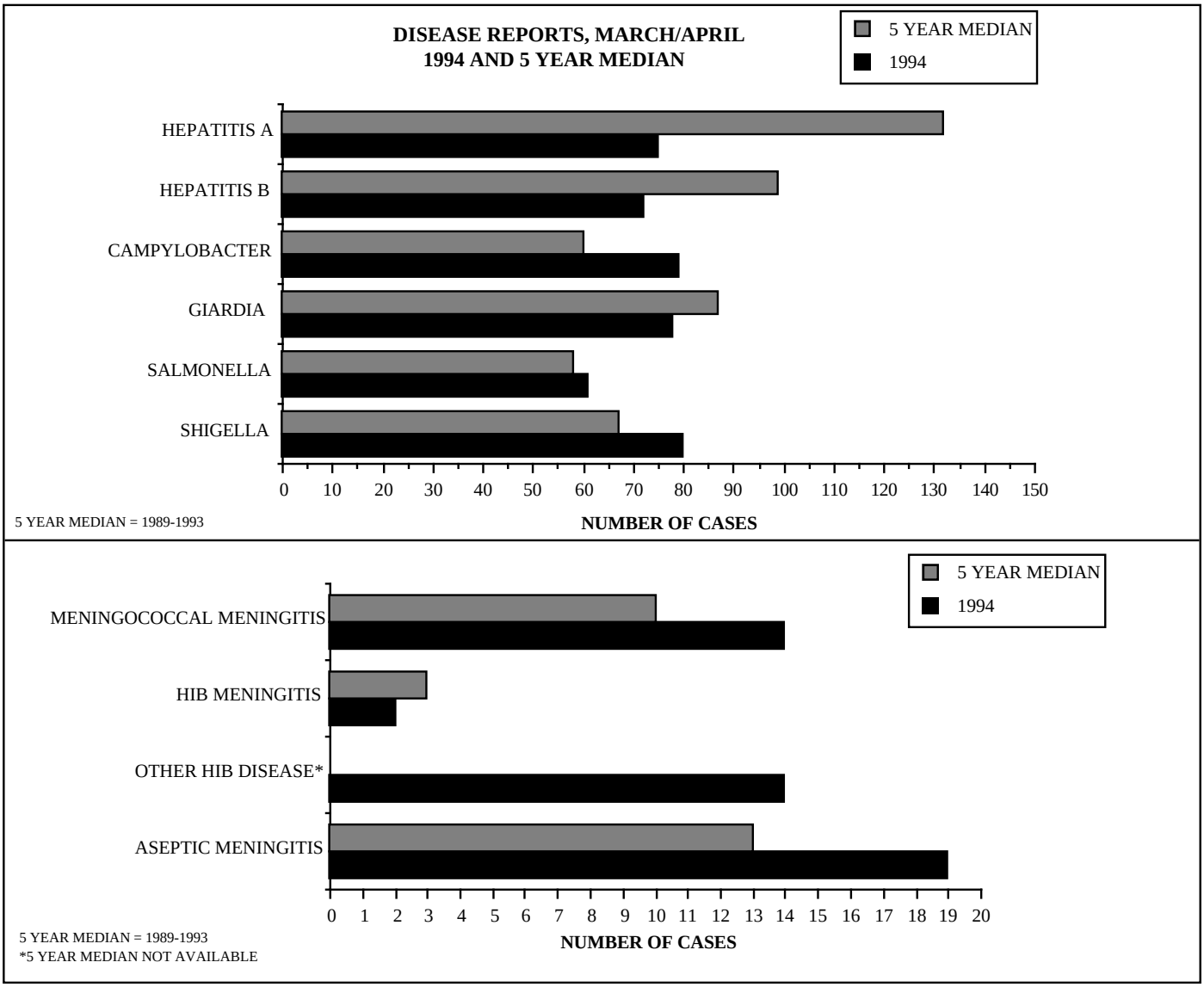
Outbreaks

Foodborne - 3
Waterborne
Nosocomial - 3
Pediculosis
Scabies
Other
- Shigella - 1
- AGI - 1
- Pneumonia - 1

*Reporting Period Beginning February 27, Ending April 30, 1994.
**Totals do not include KC, SLC, SLCo, or Springfield
***State and Federal Institutions
Data not available

Due to data editing, totals may change.

TEAR OUT FOR FUTURE REFERENCE



VIRAL HEPATITIS
Hepatitis A incidence continues to fall as it returns to pre-1992 levels. It decreased by 75.2%, from 290 cases during March/April 1993 to 72 cases during March/April 1994. This is 45.5% below the five year bimonthly median of 132 cases. Hepatitis B cases decreased by 34.2%, from 114 in 1993 to 75 in 1994. This was 24.2% less than the five year bimonthly median of 99 cases.

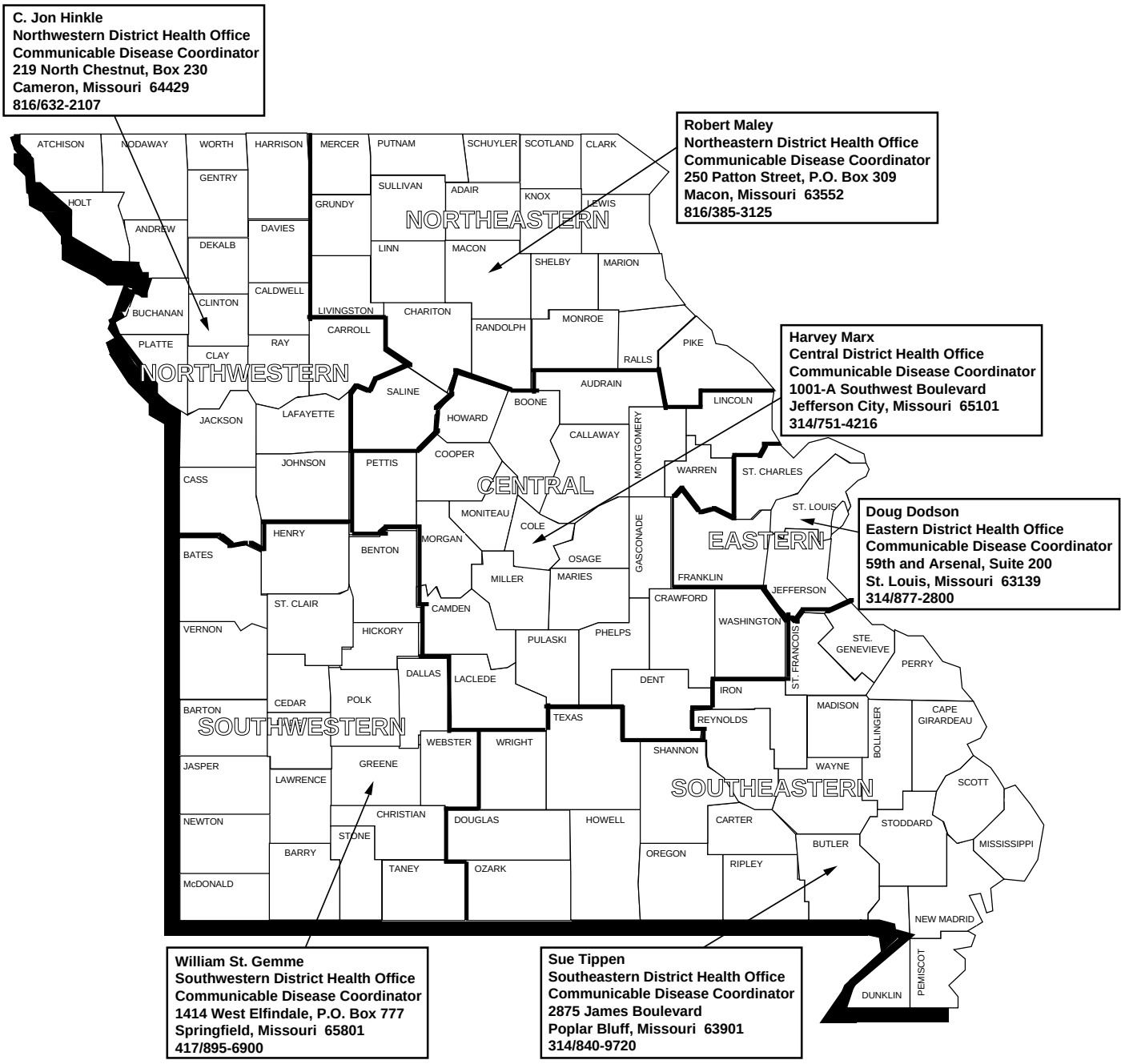
ENTERICS
Campylobacter has risen 31.7% to 79 cases during the bimonthly period from 60 in 1993, which is also the five year median. Salmonella, at 61 cases for the 1994 period, is down 6.2% from 65 cases in 1993 and up 5.2% from the five year median of 58 cases. Shigellosis decreased 22.3% from 103 cases in 1993 to 80 cases in 1994. This is 19.4% higher than the five year median of 67 cases.

PARASITES
There were 78 giardia cases reported in 1994, down 8.2% from 85 cases in 1993. This is 10.3% lower than the five year median of 87 cases.

MENINGITIS
Aseptic meningitis increased by 46.2% to 19 cases in 1994 from 13 cases in 1993, also the five year median. Meningococcal meningitis increased 40.0% to 14 cases in 1994 from 10 cases in 1993, also the five year median.

HIB DISEASE
Two cases of Hib meningitis were reported for the 1994 period, an increase of 100.0% from one case in 1993. This is 33.3% lower than the five year median of 3 cases. Reporting of other invasive Hib disease decreased 22.3%, from 18 cases in 1993 to 14 cases in 1994. There is no five year median available for other invasive Hib disease.

Missouri Department of Health
Bureau of Communicable Disease Control



This map depicts the division of counties into Department of Health districts and gives the names of the Communicable Disease Coordinators. Feel free to contact your district office regarding public health concerns.

Missouri Morbidity and Mortality Reports of Selected Communicable Diseases - 15 Year Report

	1993	1992	1991	1990	1989	1988	1987	1986	1985	1984	1983	1982	1981	1980	1979
AIDS	1664	662	656	599	481	403	239	91	52	28	6	1	-	-	-
Amebiasis	54	23	25	26	19	30	27	26	28	44	45	11	28	15	29
Brucellosis	0	0	3	1	2	4	14	4	12	7	4	4	4	3	6
Campylobacter	616	614	602	547	473	441	260	281	304	260	166	115	78	49	-
Chickenpox	9609	10009	7678	10591	9086	11350	8595	5093	2474	2565	408	637	880	2331	3510
Chlamydia	11625	11907	10643	11151	8151	6239	2944	1532	412	9	-	-	-	-	-
Encephalitis, Inf.	26	16	22	12	6	8	11	13	12	11	28	16	10	13	16
Giardiasis	770	739	790	878	859	654	690	516	458	462	216	235	113	77	72
Gonorrhea	13147	14887	17450	20012	21053	17241	16491	19029	20023	20042	20750	21269	22249	21640	21395
Haemophilus influenzae type B															
Meningitis	12	22	42	88	106	138	131	172	108	104	86	66	-	-	-
Other Invasive	123	59	39	57	-	-	-	-	-	-	-	-	-	-	-
Hepatitis A	1443	1500	653	619	810	897	560	126	98	138	123	204	282	254	392
Hepatitis B	585	535	549	633	704	639	460	420	359	297	365	297	307	205	267
Non A, Non B	25	27	31	42	53	50	46	39	42	18	33	24	(Included in Hepatitis, Unspecified)		
Unspecified	19	9	15	19	13	21	21	15	24	46	87	95	214	176	189
Influenza (confirmed)	272	111	462	220	293	148	69	78	61	39	140	153	225	-	-
Lyme Disease	108	150	207	205	108	-	-	-	-	-	-	-	-	-	-
Malaria	9	12	9	13	13	6	8	12	5	8	4	10	4	16	6
Meningitis, Asep.	275	272	277	246	223	124	163	172	156	95	277	156	178	116	130
Meningitis, Mening.	34	32	37	31	21	33	35	40	46	53	55	40	45	42	38
Meningitis, Other	78	43	62	66	64	64	75	123	47	51	276	156	122	127	94
Mumps	46	39	40	62	87	68	38	23	18	11	21	13	40	103	203
Pertussis	144	120	83	116	141	25	46	32	35	23	24	17	24	30	24
Polio, all forms	0	0	0	0	0	1	0	0	1	0	2	0	1	0	1
Rabies, Animal	35	37	28	30	62	36	59	75	59	70	96	123	243	379	307
RMSF	20	24	25	36	48	54	26	25	10	14	14	10	23	31	31
Rubella	1	1	5	3	4	0	0	1	7	0	0	38	2	45	73
Rubeola	1	0	1	103	671	65	190	32	5	6	1	2	1	67	436
Salmonellosis	529	426	616	723	676	772	660	728	690	617	602	571	700	589	602
Shigellosis	674	742	259	284	411	607	471	89	143	244	264	67	268	129	258
Syphilis, Total	2499	1940	926	598	388	473	328	494	578	712	801	1069	1397	1051	896
Primary & Second.	1354	1167	572	272	162	154	90	110	133	186	145	296	394	163	139
Tetanus	1	1	1	0	4	1	1	2	3	6	1	1	1	2	1
Tuberculosis	256	245	254	312	278	275	339	338	311	354	399	390	432	466	500
Tularemia	17	34	44	33	39	45	58	32	35	40	51	27	28	26	21
Typhoid Fever	2	3	2	4	2	3	7	6	6	6	10	4	9	20	8
Yersinia enterocolitica	26	37	48	32	36	30	10	6	2	3	1	-	-	-	-

Bureau of Communicable Disease Control 1993 Annual Report

(continued from page 3)

Haemophilus influenzae type b Disease

Reported cases of Hib meningitis decreased 45.5 percent, from 22 cases in 1992 to 12 cases in 1993, despite intensive, laboratory-based surveillance for cases by the CDC funded Invasive Bacterial Disease Surveillance Project that started in January 1992. The 1993 total is 86.4 percent lower than the five-year median of 88 cases. See Figure 7. Reported cases of other invasive (non-meningitis) Hib disease increased by 108.5 percent, from 59 cases in 1992 to 123 cases in 1993. See Figure 7. This increase appears best explained by the detection of additional cases that would have remained undetected had the surveillance project not been in place. Since other invasive Hib disease has been reportable only since 1990, there is no five-year median.

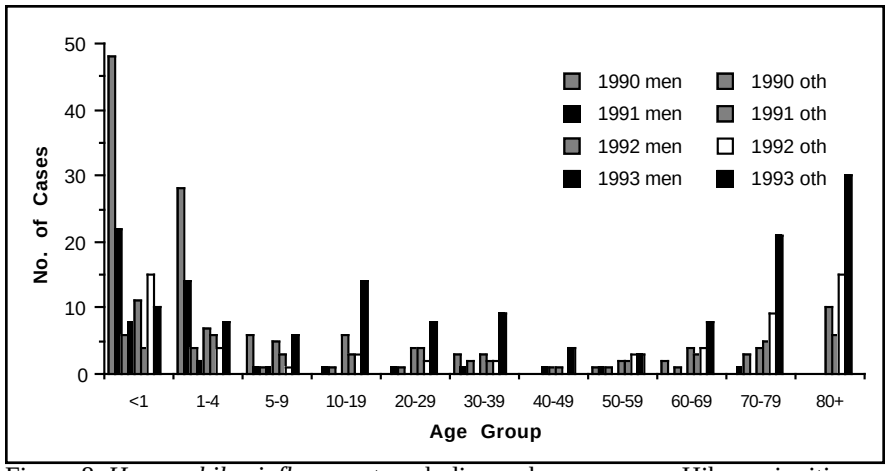


Figure 8. *Haemophilus influenzae* type b disease by age group, Hib meningitis and Other invasive Hib disease, Missouri, 1990–93.

The age distribution of Hib disease has changed with the widespread use of Hib vaccines, and meningitis is no longer the most common expression of the disease. There has been a reduction in the number of cases of Hib meningitis in children under five, but little change in the number of reports of other types of invasive Hib disease. There are few reports of Hib meningitis in people 10 years of age and older, however, the number of invasive Hib disease cases reported in older individuals, particularly those over 50, has increased due to intensive laboratory surveillance. See Figure 8.

REFERENCE:

1. CDC. *E coli* O157:H7–what the clinical microbiologist should know. March 1994.

1993–94 Influenza Summary

(continued from page 12)

Resources reported that pneumonia and influenza deaths in the state increased by 15 percent and replaced accidents as the fifth leading cause of deaths in Missouri during 1993.

Because of budgetary and procedural constraints, the State Public Health Laboratory cut back on the number of sites from which they would accept cultures. This was accomplished by selecting two locations from each district to serve as surveillance sites. The laboratory accepted cultures from other sites when outbreaks or increased incidence was reported. This procedure reduced the number of cultures performed by 30 percent compared to last season and by 59 percent when compared to the average of the last four seasons. Figure 4 shows laboratory confirmed influenza cases by county of residence.

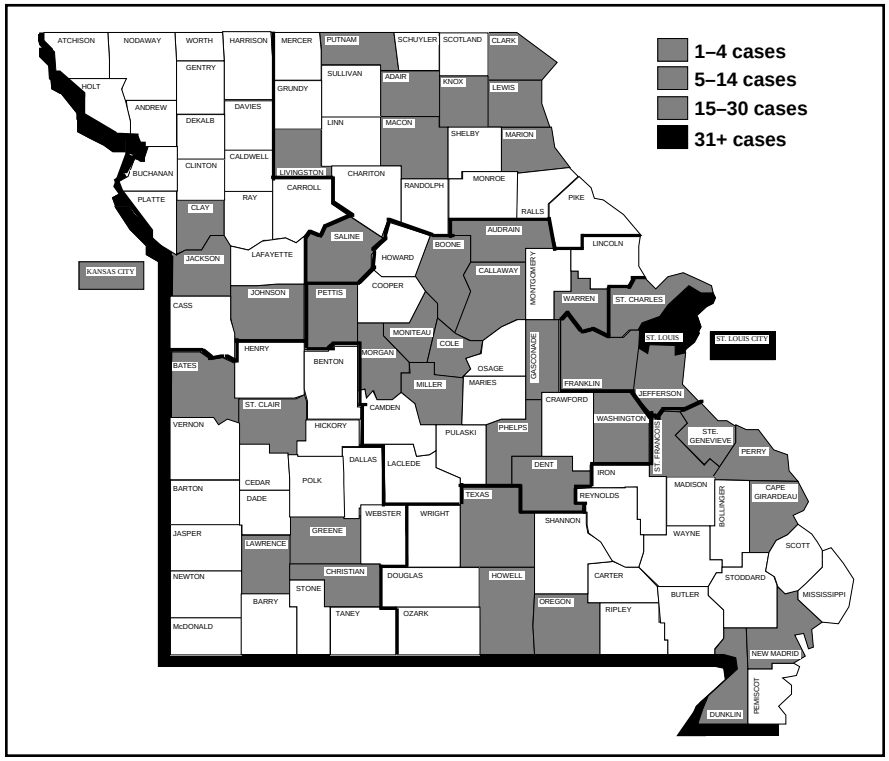


Figure 4. Laboratory confirmed influenza cases by county of residence, Missouri, 93/94 season.

1993 Rabies Summary

F.T. Satalowich, D.V.M., M.S.P.H.
Bureau of Veterinary Public Health

Emerging pathogens, or more correctly **re-emerging** pathogens, are capturing minds and emotions of scientists and media personnel alike. The rabies raccoon epizootic in the northeastern United States is a typical example of the public hysteria that can prevail. See Figure 1. While the epizootic is indeed real, the reality of living with endemic rabies, which Missouri and the midwest has done for decades, is not. From 1991–93, raccoon rabies has increased by 92 percent, from 3,079 to 5,912 cases. New York alone tested 4,463 raccoon specimens with 2,369 (53%) being positive. The number of persons receiving the post-exposure rabies treatment in New York increased from 1,125 in 1992 to 2,905 in 1993.

Nationally, the number of rabies cases rose about ten percent from 1992–93, for a total of 9,498 rabies cases in 1993. The majority of rabies cases are in wild animals (92%), with the raccoons epizootic dominating the picture with 5,912 cases. Skunks, which in the past accounted for most cases, only accounted for 1,640 cases. Because of their ecological association with raccoons, there were 59 cases of groundhog rabies. Largely due to the adaptation of wild dog rabies to coyotes in Texas, 74 cases of coyote rabies were reported.

Bats accounted for about eight percent of wildlife rabies with 759 cases in 1993, a 17 percent increase from 1992. Domestic animal rabies only represented eight percent (606) of the total rabies cases, with 130 dogs, a 29 percent decrease, and 291 cats. Although the number of cat rabies cases stayed about the same, it depicts the slow reaction to the need to vaccinate cats. Cattle rabies (130 cases) decreased by 29 percent. Cattle are normally exposed by skunks, thus the increase in raccoon rabies, especially urban raccoon rabies, has not affected cattle.

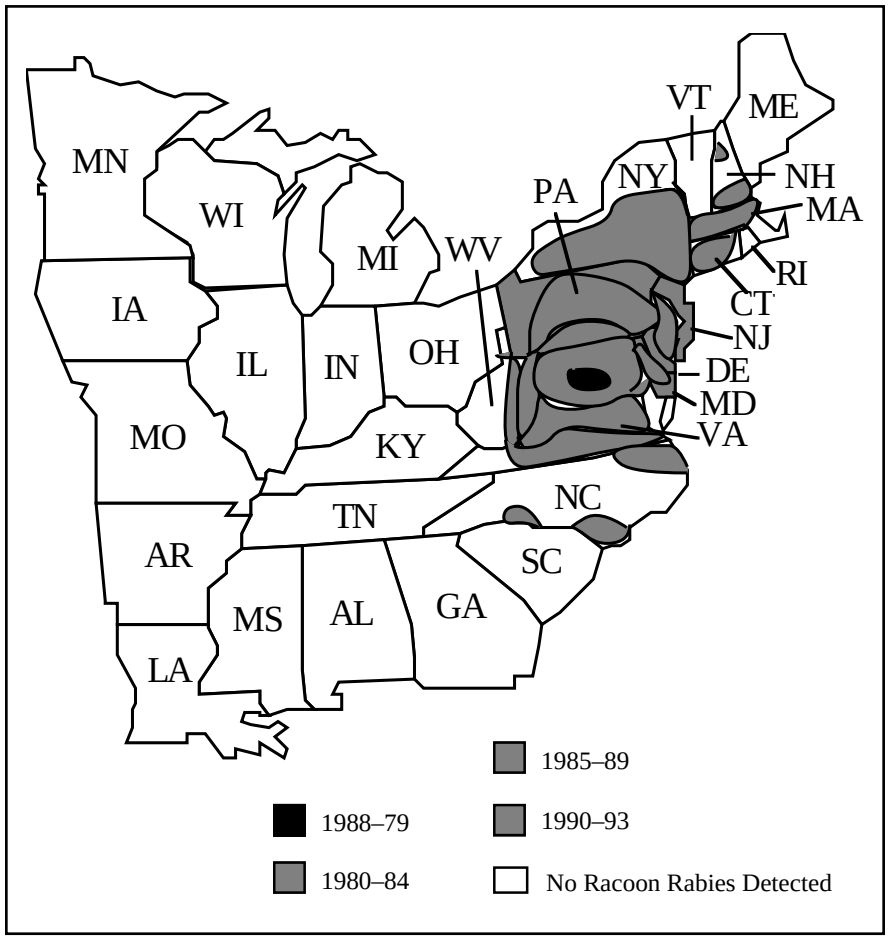


Figure 1. Raccoon rabies epizootic, United States, 1988–93.

The midwest, and especially Missouri, is at an all time low for rabies activity. Missouri had 36 and 35 rabies cases in 1992 and 1993 respectfully. Iowa had a 45 percent decrease in animal rabies, from 175 cases in 1992 to 79 cases in 1993. Illinois had only 23 animal rabies cases, 19 bats, 3 skunks and 1 dog.

Of the 35 Missouri cases in 1993, 89 percent were in wild animals, 21 percent in bats and 10 percent in skunks. Eleven percent of the cases were in domestic animals, one each in a bovine, horse, dog and cat. Oddly enough the data shows that even at this low incidence, all domestic species can be affected by wildlife rabies. Location of the domestic animal cases further proves how endemic rabies is throughout Missouri. The bovine case was reported from Clinton county in northwest Missouri, the equine case from Franklin county in eastern Missouri, the canine case from Madison county in southeastern Missouri and the feline case from Howell county in south central Missouri on the Arkansas border. All these represented the only rabies case reported from that county. See Figure 2. While this distribution illustrates the endemicity of rabies throughout Missouri, it also depicts the inability of our local surveillance system to pick up rabies in the natural wild reservoir prior to its occurrence in the domestic species.

Historically, the rabies laboratories in the state have conducted some 2,600–2,700 rabies examinations per year. Be-

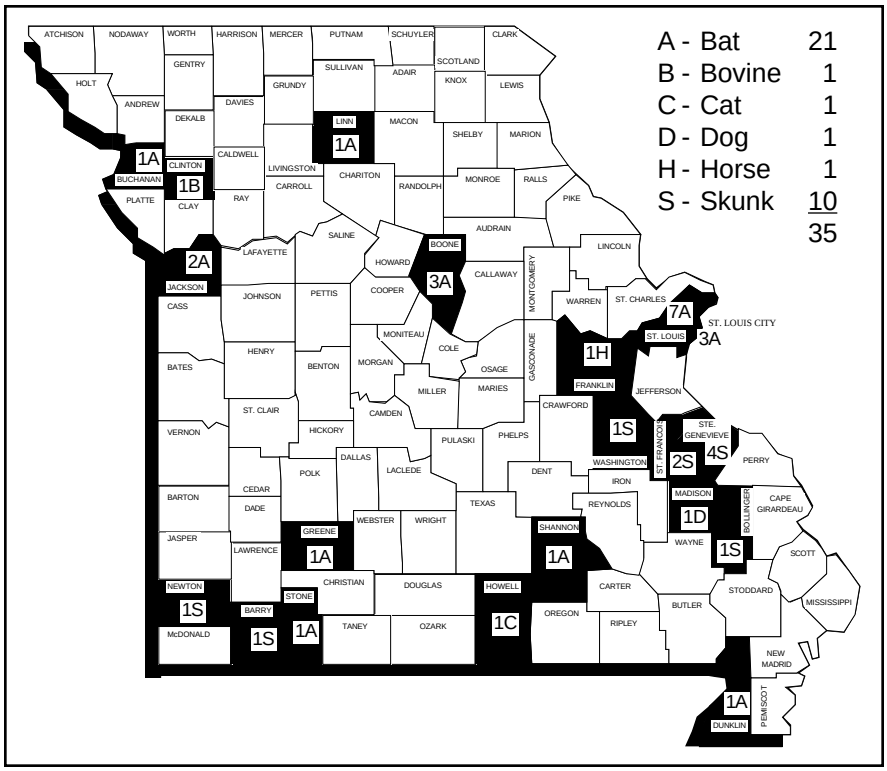


Figure 2. Animal rabies cases, Missouri, 1993.

Table 1. Number of animal rabies specimens examined and positive, Missouri, 1988–93

	1988	1989	1990	1991	1992	1993
Total Examined	2,472	2,437	2,324	2,371	2,708	2,145
Total Positive	36	62	30	28	37	35
Percent Positive	1.5%	2.5%	1.3%	1.2%	1.4%	1.6%

cause of society's increased involvement with exotic animals, the percent of rabies specimens that are true indicators of the prevalence of rabies in the reservoir or spill-over species has decreased. Thus, the passive rabies surveillance being used to assess the incidence of rabies in wildlife species is being impaired.

A number of tactics are being employed to preserve the validity of the passive surveillance system. First, rabies laboratory specimens are only being accepted from local health departments or health-care providers. Secondly, educational means are being utilized to inform submitters of specimens to use proper judgment in the selection of epidemiologically-appropriate specimens. By the utilization of these methods, the number of rabies specimens has dropped to 2,300–2,400 per year during the past five years, and was down to 2,145 specimens in 1993. See Table 1.

While appropriate rabies specimens are important, significant numbers of specimens are also important to provide a true picture of rabies prevalence in Missouri or a specific county. A long established goal of a minimum of three appropriate specimens per county per month is still in effect. Specimens from animals causing human exposure are always accepted if deemed appropriate by the health-care provider. Based on over 50 years of testing in Missouri and by CDC without a single positive result, specimens from mice, rats, guinea pigs, hamsters and gerbils are not accepted for testing.

With an appropriate selection of specimens, a statistically-valid observation can be made. The percent of total positives usually increases significantly one year before the total number of rabies cases increases. The key ingredients are an appropriate number of specimens and what constitutes a significant positive increase.

One of the obstacles in getting appropriate rabies specimens to the laboratory is the transport of that specimen from remote areas where a true interaction of domestic and wild animals occurs. Each county health administrator and submitting health-care provider should research the various avenues of transport and establish the most cost efficient and effective means prior to the necessity for utilization. The Department of Health has recently obtained funds to establish a laboratory courier service. Local county health departments should be aware of this service and how health-care providers in their area can access it.

There were three cases of human rabies in the United States in 1993. The California case was acquired from a dog in Mexico. The other two cases occurred in Texas and New York and were acquired from silver-tailed bats. The raccoon epizootic, which now transcends over 15 years, has not produced a single case of human rabies. The cardinal rules of rabies control are vaccination of dogs and cats to serve as a buffer zone between the wildlife reservoir and domestic animals, the control of stray animals and avoiding contact with wild animals. This continues to prevent human rabies cases and serves as the most cost-effective method of rabies control. Increased contact with wild and stray animals has resulted in increased administration of human post-exposure rabies treatment and the examination of suspect rabies specimens. Whether these costs have approached the justification for wildlife rabies control with oral vaccine has not been determined.

Tick-Borne Disease Summary - 1993

F. T. Satalowich, D.V.M., M.S.P.H.
Bureau of Veterinary Public Health

Rocky Mountain Spotted Fever

Rocky Mountain Spotted Fever (RMSF) is characterized by sudden onset of symptoms including headache, conjunctivitis, peripheral and periorbital edema, chills, fever lasting two to three weeks, myalgia and a maculopapular rash usually appearing on the second to sixth day. The rash is the most characteristic and helpful diagnostic sign. It usually appears first on the wrists and ankles and may include the palms and soles, spreading centripetally to the rest of the body. If treatment is delayed, petechiae and purpuric skin lesions are common. Health professionals are encouraged to investigate the possibility of tick exposure when diagnosing illnesses in patients presenting with these symptoms. The infectious agent of RMSF is *Rickettsia rickettsii*. Even though dogs, rodents and other small animals may harbor the rickettsiae, the principle vector and reservoir is the tick.

Ninety percent of the thousand rickettsial diseases that occur annually in the United States are RMSF. During the 1980's, approximately 50 deaths per year were attributed to RMSF. The total number of cases nationally has increased since the 1960's and peaked in 1981. While the national incidence, and especially the incidence in southeastern states plateaued or decreased, the incidence of RMSF in Arkansas, Oklahoma and Texas increased between 1981–83 by 107 percent. This was followed by a 50 percent decrease in those states in 1984–85. The majority of cases (83%) in those states occurred between April and August and 67 percent of cases were in males. The case fatality ratio was 4.7 percent, with rates being higher in blacks and elderly. The endemic foci of RMSF that exists in Arkansas, Oklahoma and Texas has an annual incidence trend that differs from the rest of the nation.

Missouri does not totally follow either trend. See Figure 1. From 1982–85, Missouri averaged 12 cases per year. The four years prior, 1978–81, Missouri averaged 28.5 cases per year. The number of cases from 1986–88 has increased yearly with 25, 26 and 54 cases occurring in those respective years. From 1989–93, the number of cases has decreased progressively from 48 cases in 1989 to 20 cases in 1993. Missouri's highest number of cases occurred in 1988, seven years after the nation experienced its highest number of cases in 1981. This increased number of cases is due to the normal cycling of disease. Better diagnostic procedures and surveillance also play a role. Missouri had one death reported from RMSF in each of 1988, 1990 and 1992. There has also been an increased number of cases and deaths in dogs in recent years. Figure 2 shows the distribution of RMSF cases by county in Missouri in 1993.

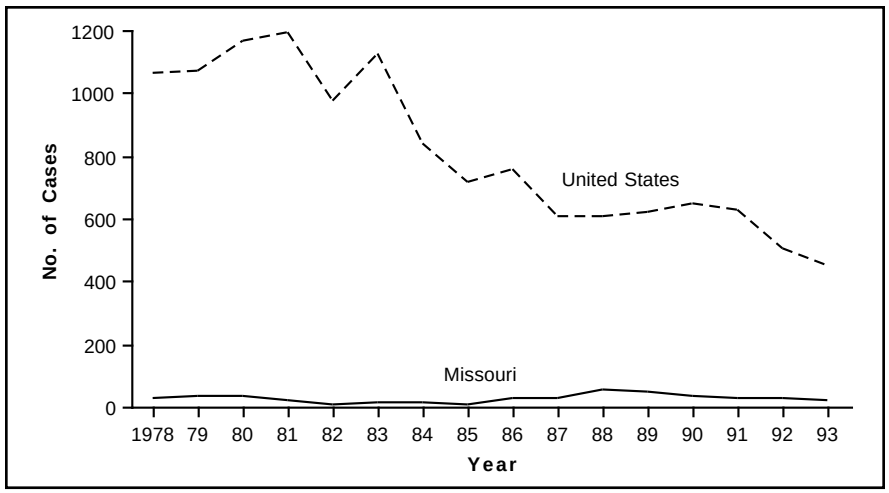


Figure 1. Rocky Mountain spotted fever cases by year of report, Missouri and United States, 1978–93.

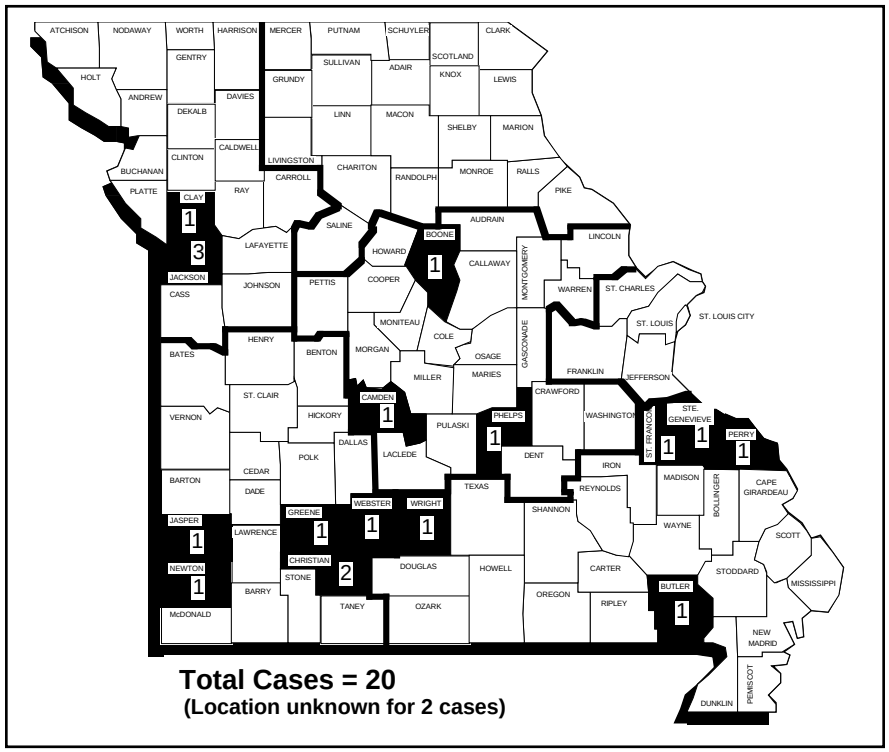


Figure 2. Rocky Mountain spotted fever cases by county, Missouri, 1993.

Tularemia

Tularemia is a disease of man and animals caused by the bacteria *Francisella tularensis*. Tularemia is also called rabbit fever and deerfly fever. Tularemia is enzootic in animals throughout the continental United States and in most areas of the world between 30 to 71 degrees north latitude. Based on biogeographic epidemiology, Missouri lies in one of the two recognized tularemia regions in the North American continent. This region, called the Ozark Plateau, encompasses portions of Missouri, Arkansas, Oklahoma and Kansas.

Since 1983, Missouri has had a total of 428 cases of tularemia reported, or approximately an average of 39 cases per year. See Figure 3. The number of reported cases has been increasing with 51, 58, 45 and 44 cases occurring in 1983, 1987, 1988 and 1991, respectfully. Missouri led the nation in the total number of cases for those years. In 1981, 1984, 1985 and 1986, Missouri ranked number two in reported cases, behind either Arkansas or Oklahoma.

Most tularemia cases in Missouri occur south of the Missouri River. Figure 4 shows the distribution of cases by county in Missouri in 1993.

Ehrlichiosis

Erhlichiosis is an acute febrile illness of humans caused by *Ehrlichia chaffeensis* and thought to be transmitted by the brown dog tick, *Rhipicephalus sanguineus*. As with other tick-borne diseases, it has an acute onset with flu-like symptoms including headache, myalgia, anorexia, nausea and, in some instances, a rash. Clinical laboratory abnormalities include leukopenia, thrombocytopenia and elevated levels of hepatic aminotransferase.

Human ehrlichiosis infections have been recognized in the United States since 1985 when a case was identified in an Arkansas resident. Since that time, 320 additional cases have been reported from 24 states with nine fatalities. Missouri has reported the highest number of cases

(continued on page 22)

Figure 3. Tularemia cases by year of report, Missouri, 1983–93.

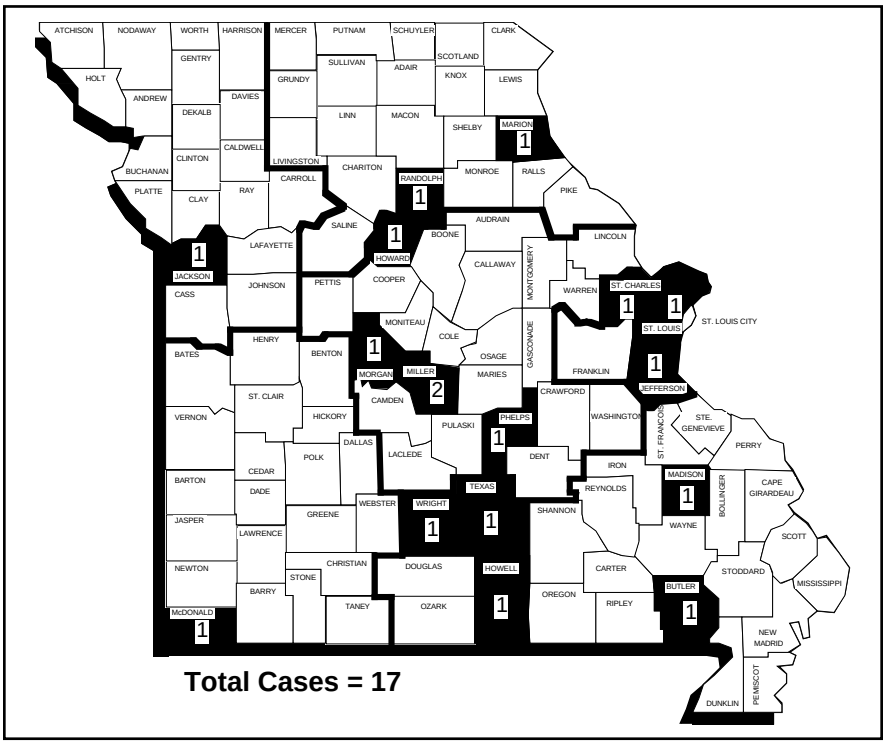


Figure 4. Tularemia cases by county, Missouri, 1993.

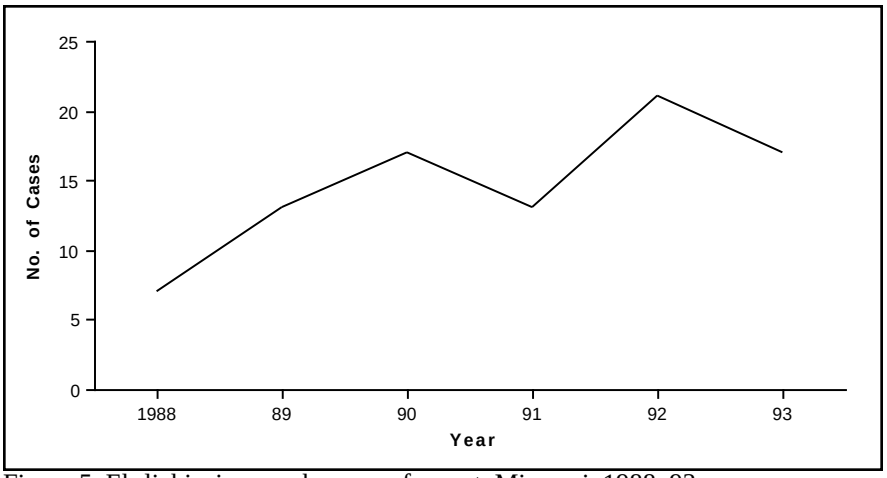


Figure 5. Ehrlichiosis cases by year of report, Missouri, 1988–93.

(continued from page 21)
since 1988 with a total of 88 cases, or an average of 15 cases per year. See Figure 5. Figure 6 shows the distribution of ehrlichiosis cases by county in Missouri in 1993.

Lyme Disease

A bacterial illness transmitted by ticks to wildlife and man, Lyme disease has become the most commonly reported vector-borne disease in the United States with as many as 90 percent of all cases being reported from the northeastern United States. The tick most commonly reported as the vector for Lyme disease is *Ixodes scapularis* (formerly *Ixodes dammini*). *I. scapularis* is not common in Missouri. Other possible vectors include *Amblyomma americanum* (the Lone Star tick) and *Dermacenter variabilis* (the dog tick).

The number of reported Lyme disease cases increased dramatically after it was designated a reportable disease in Missouri in June 1989, then declined during 1992–93. See Table 1. There were 108 cases reported in 1993 which met the case criteria set by the Centers for Disease Control and Prevention and the Council of State and Territorial Epidemiologists. This included five cases who were exposed outside of the state. Figure 7 shows the distribution of lyme disease cases by county in Missouri in 1993.

Table 1. Lyme disease cases in Missouri

Year	Suspects Reported	Definite Exposed in Missouri	Definite Exposed Elsewhere
1983	1	0	0
1984	10	0	2
1985	6	1	0
1986	5	0	1
1987	30	4	0
1988	38	4	1
1989	213	106	2
1990	420	194	11
1991	415	200	7
1992	466	142	8
1993	232	103	5
Total	**1,836**	**754**	**37**

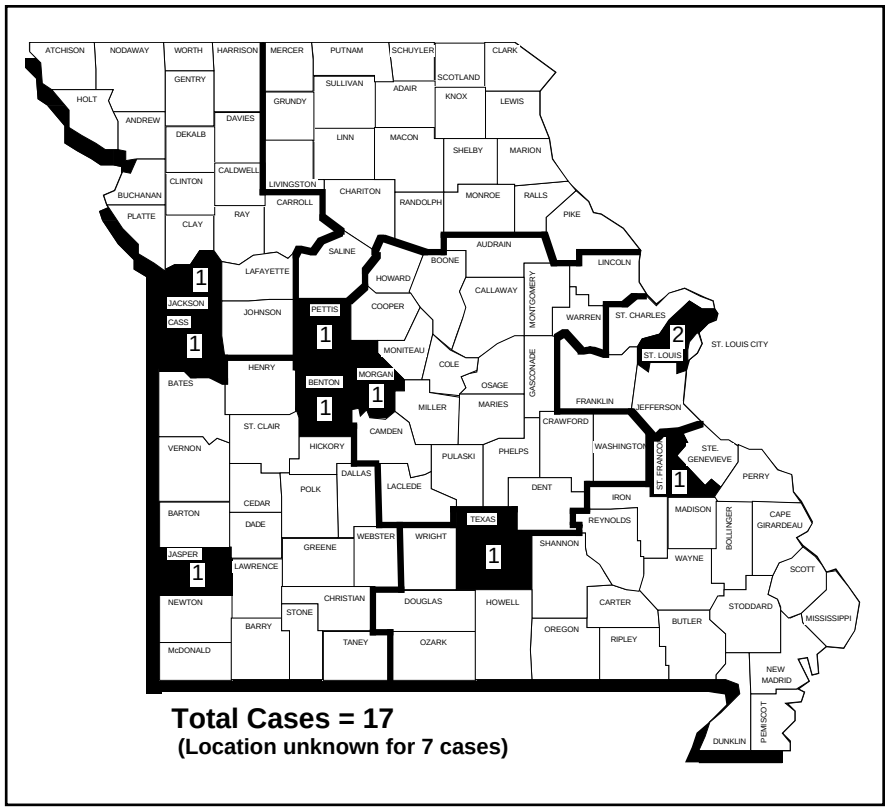


Figure 6. Ehrlichiosis cases by county, Missouri, 1993.

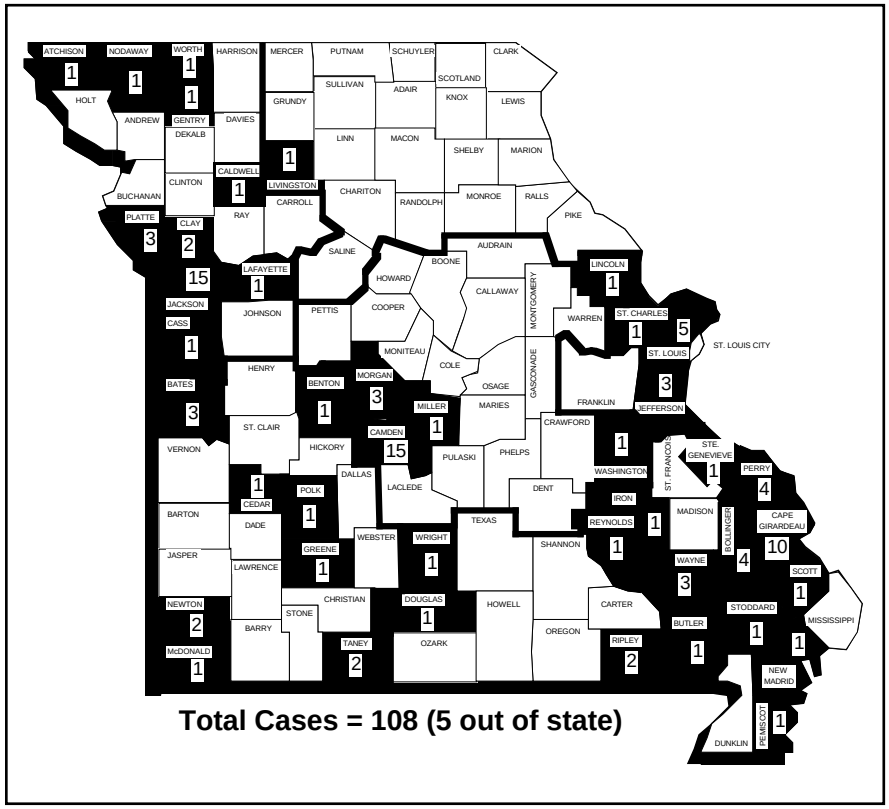


Figure 7. Lyme cases by county, Missouri, 1993.

Vaccine-Preventable Diseases - 1993

Mary Ann Harder, M.S.
Bureau of Immunization

One hundred forty-four cases of pertussis were reported in Missouri during 1993, an increase of 24 cases over the number reported in 1992, and an increase of 119 cases over 1988. See Figure 1. Of the 144 reported cases, 80 (56%) were in infants six months of age or younger. Table 1 shows the reported cases by age group and vaccination status. Cases were reported from all of Missouri's seven health districts. See Figure 2. Sixty-nine percent of the cases occurred in the four-month period from June through September. Figure 3 shows reported cases by month of onset.

Nationwide, pertussis is at a 27-year high, with more than 6,000 cases of whooping cough, and at least eight deaths, reported in the United States in 1993.[1]

During 1993, one case of measles was reported, whereas 1992 was a measles-free year. This is in contrast to 671 reported cases in 1989, the year the Centers for Disease Control and Prevention Advisory Committee on Immunization Practices (ACIP) initiated the recommendation for two-dose measles protection. See Figure 1.

One case of rubella was reported, the same as in 1992. Forty-six cases of mumps were reported, an increase of seven cases over the previous year. A single case of tetanus was reported in a 26-year-old man who had stepped on a nail in a yard.

During 1993, the Missouri General Assembly expanded the state's prenatal screening law to include a screen for hepatitis B as part of the initial prenatal panel. Any pregnant woman found to be HBsAg-positive may have further screening, along with hepatitis B vaccine for her contacts, provided by the Bureau of Immunization at no charge. Obstetricians and their delivery hospitals are urged to work together to ensure

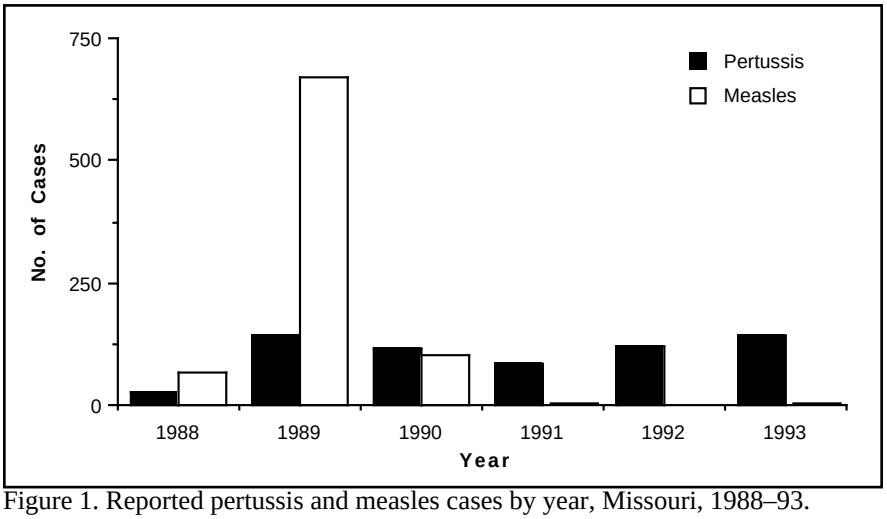


Figure 1. Reported pertussis and measles cases by year, Missouri, 1988–93.

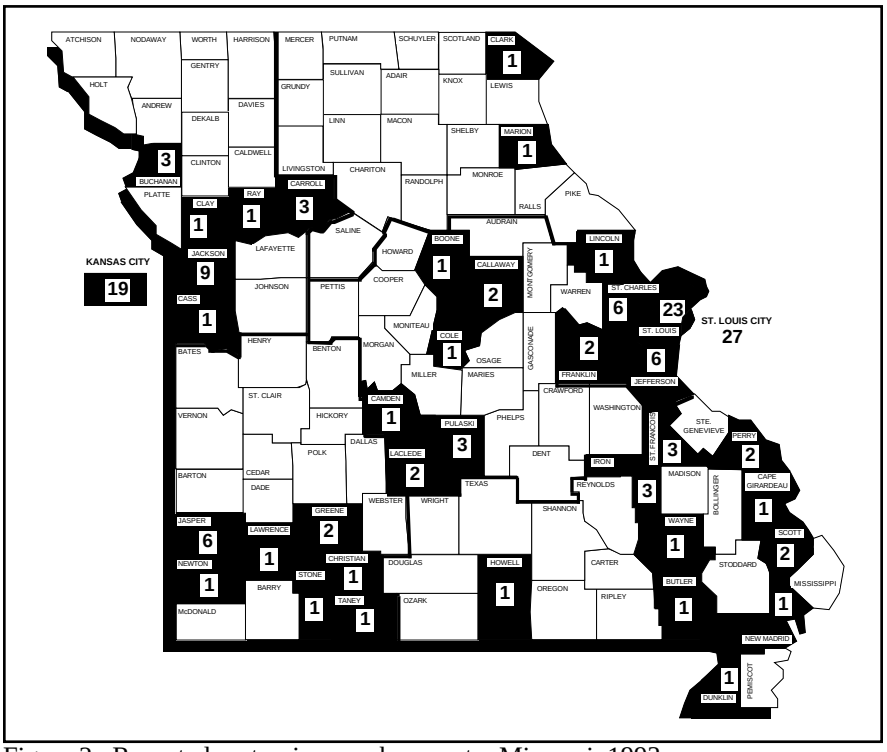


Figure 2. Reported pertussis cases by county, Missouri, 1993.

that every woman's HBsAg status (positive or negative) is documented in her delivery chart.

All neonates should be given their first dose of hepatitis B vaccine as soon as they are physiologically stable after delivery. If the HBsAg status of the mother is unknown, the infant should be given hepatitis B vaccine when physiologically stable and within 12 hours of birth in a dose appropriate for infants born to HBsAg-positive mothers. The mother in this situation should be immediately tested for the presence of HBsAg. If she is found to be HBsAg-positive, the infant should receive the additional protection of hepatitis B immune globulin (HBIG) as soon as possible and within seven days of birth, although the efficacy of HBIG administered after 48 hours of age is not known.

(continued on page 26)

Tuberculosis Summary - 1993

Dan Ruggiero
Bureau of Tuberculosis Control

During the period from 1986 through 1992, the United States experienced a continuing increase in tuberculosis morbidity. However, in 1993, the number of newly reported cases dropped to 25,313, a decrease of 5.1 percent from 1992. This decrease reflects the increased awareness, resources and emphasis placed on prevention and control measures in the past few years.

From 1985 until 1993, 64,000 more cases of tuberculosis were seen nationwide than would have been expected based on previous trends. These excess cases have largely been attributed to HIV-infected persons who were also infected with *Mycobacterium tuberculosis*. Increased tuberculosis morbidity occurred mainly in those geographic areas, and in those demographic groups**,** which had large numbers of AIDS cases.

In contrast to these national trends, Missouri experienced a decline in the number of reported tuberculosis cases from 1986 to 1992. However, in 1993, 256 cases of the disease were reported, which represented a 4.5 percent increase over the 245 cases reported in 1992. See Figure 1.

The number of tuberculosis cases reported in Missouri in 1993 varied by geographic area. Missouri's outstate areas continued to account for the majority of reported cases with 131 (51%). The urban centers of the state, St. Louis City, St. Louis County, Kansas City and Springfield, accounted for the remaining 125 cases (49%). St. Louis City, St. Louis County and Springfield showed decreases of 2.3 percent, 7.7 percent and 47.1 percent, respectively, in the number of reported cases for 1993 as compared to 1992**.** However, this trend was reversed in Kansas City, which experienced an increase of 54.2 percent, or 13 more cases**,** over the number reported in 1992. See Figure 2**.**

Four health districts showed increases in the number of reported cases of tuber-

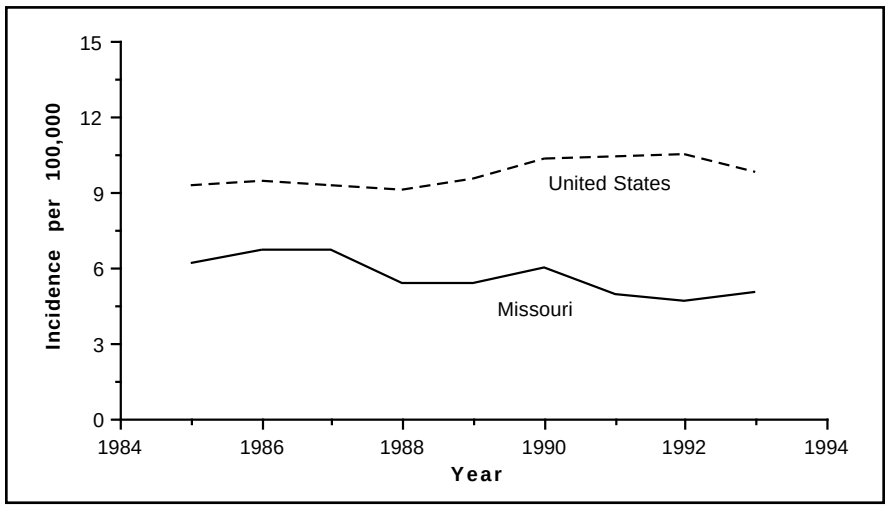


Figure 1. Tuberculosis case rates per 100,000 population, Missouri and United States, 1985–93.

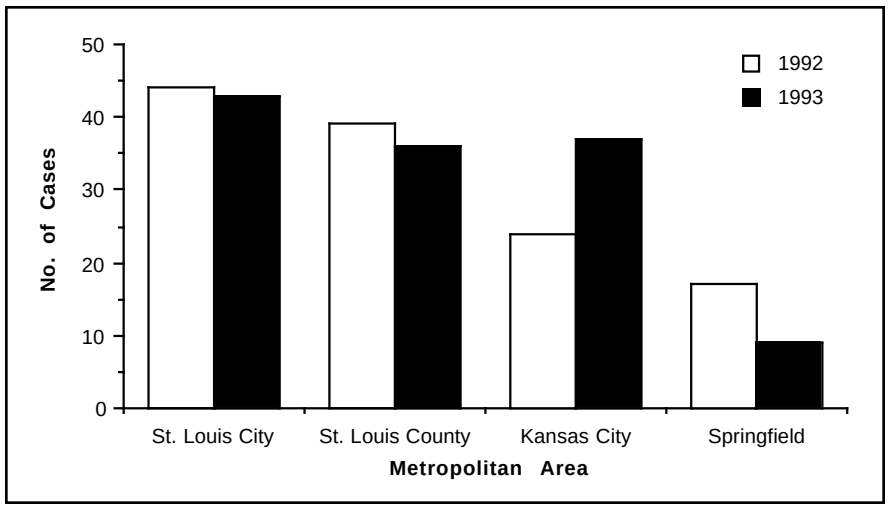


Figure 2. Tuberculosis cases by metropolitan area, Missouri, 1992–93.

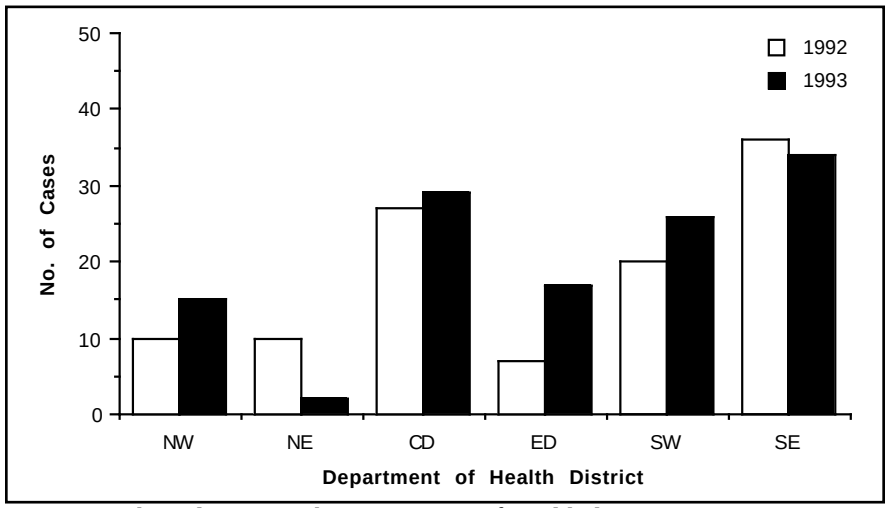


Figure 3. Tuberculosis cases by Department of Health district, Missouri, 1992–93.

culosis during 1993, as compared to 1992. The Eastern District experienced the largest increase (143%) or ten more cases than the seven reported in 1992. Increases in reported cases were also noted in the Northwestern District with 15 cases (+50%), the Southwestern District with 26 cases (+30%) and the Central District with 29 cases (+7%). Two districts showed a decrease in the number of cases reported in 1993**.** The Northeastern District had two cases (-80%) and the Southeastern District had 34 cases (-5%). See Figure 3. A decrease was also noted in correctional facilities with eight cases (-27%).

The elderly continue to comprise a large percentage of the tuberculosis cases in Missouri. Individuals age 65 and older accounted for 96 (37.5%) of the 256 cases reported during 1993, an increase over 1992, when 91 (37.1%) of the 245 reported cases were in this age group. An increasing percentage of cases was also noted during 1993 in two of the three age groups under 25 years. Compared with 1992, the percentage in the 0–4 year age group remained the same at 3.9 percent of the total cases, while the 5–14 age group experienced a 450 percent increase in reported cases, and the 15–24 age group experienced an 87.5 percent increase**.** See Figure 4.

Tuberculosis disease varied significantly among different racial and ethnic groups during 1993, with 169 (66%) of the 256 reported cases occurring among whites, 67 (26.2%) occurring among blacks and 19 (7.4%) occurring among Asians. These 1993 figures reflect a decrease in the percentage of cases reported among blacks from 28.6 percent in 1992, and a decrease in the percentage of reported cases in the Asian population from 8.2 percent. There was one case reported among American Indians. See Figure 5**.**

Missouri's minorities have experienced a disproportionately higher incidence rate of tuberculosis than that seen in the white population. The overall incidence rate for minorities in the state during 1993 was 14.3 cases per 100,000 population, with a rate of 12.3 for blacks, 48.0

(continued on page 26)

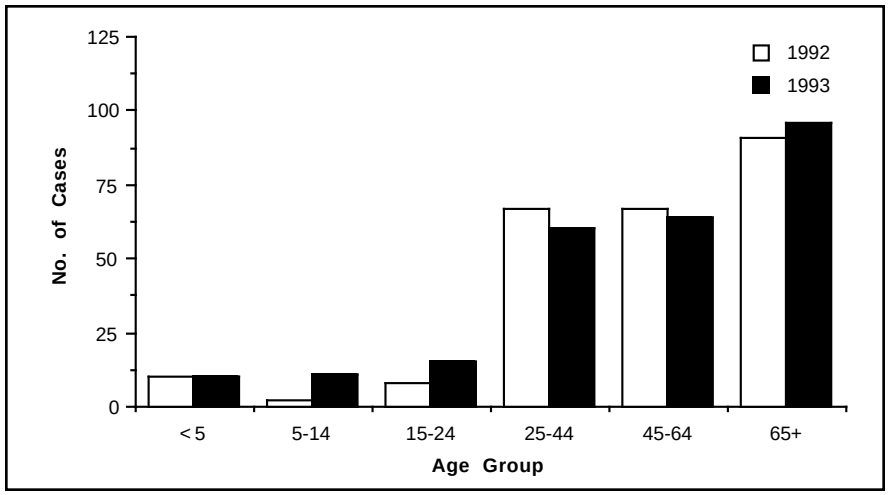


Figure 4. Tuberculosis cases by age group, Missouri, 1992–93.

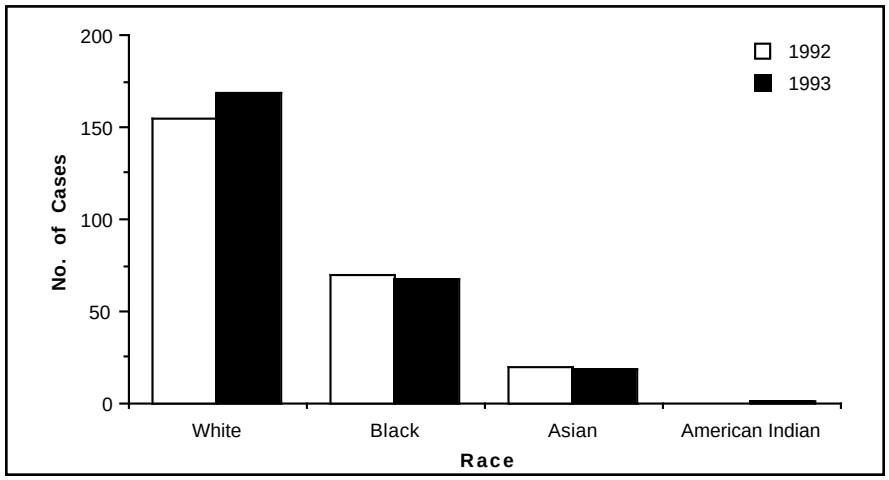


Figure 5. Tuberculosis cases by race, Missouri, 1992–93.

Table 1. TB/AIDS Cases by Place of Residence, Missouri, 1987–93

Place of Residence	1987	1988	1989	1990	1991	1992	1993	Total
St. Louis City	2	0	1	1	3	4	5	16
St. Louis County	0	2	0	1	0	1	3	7
KC-Jackson County	1	1	2	2	1	0	7	14
Springfield-Greene Co.	1	0	1	0	0	0	1	3
Outstate	2	1	2	1	0	2	2	10
Institutionalized	5	2	10	2	4	3	3	29
Total	**11**	**6**	**16**	**7**	**8**	**10**	**21**	**79**

Table 2. Drug-Resistant Tuberculosis Cases, Missouri, 1987–93

Cases with Resistance to:	1987	1988	1989	1990	1991	1992	1993	Total
One Drug	13	9	13	10	14	9	11	79
Two Drugs	2	4	7	4	0	4	3	24
Three Drugs	0	1	0	2	1	2	1	7
Four Drugs	0	0	0	2	0	0	0	2
Five Drugs	0	0	0	1	0	0	0	1
Total	**15**	**14**	**20**	**19**	**15**	**15**	**15**	**113**

(continued from page 25)
for Asians and 4.5 for American Indians. This compares to a rate of 3.8 for whites. Overall, minority populations in Missouri are nearly four times as likely to contract tuberculosis as whites, with rates exceeding that of the national rate of 9.8.

The percentage of tuberculosis cases in St. Louis City among the black population remained the same in 1993, accounting for 65.1 percent of the total cases reported. In St. Louis County, the percentage of cases among blacks increased from 28.2 percent in 1992 to 36.1 percent in 1993. In Kansas City, the percentage of cases in blacks decreased from 58.3 percent in 1992 to 35.1 percent in 1993.

Of increasing concern is the number of tuberculosis cases in children under 15 years of age. Between 1992 and 1993, the number of children under the age of 15 with tuberculosis rose from 12 to 21, an increase of 75 percent. This does not speak well of tuberculosis control efforts, since it represents the occurrence of new, active transmission of infection.

Also of concern is the increasing number of persons being reported in Missouri with both tuberculosis disease and AIDS. Of the 4,876 cases of AIDS reported among Missourians through 1993, a total of 79 individuals (1.6%) were also known to have been diagnosed with tuberculosis. The number of TB/AIDS patients has more than doubled over the past two years, from ten individuals reported in 1992 to 21 in 1993. As expected, these persons are primarily residents of the metropolitan areas, or are confined in correctional institutions. See Table 1.

Newly-arriving immigrants are another group which account for an increased incidence of tuberculosis in Missouri. Foreign-born persons made up 12.1 percent of all reported cases during 1993. This represents an increase over the percentage of foreign-born cases in both 1992 (11.8%) and 1991 (9.8%).

Although much has been written about the increase in drug-resistant disease nationally, little has been seen so far in Missouri. In 1993, there were 15 cases of drug resistance, 11 of which were resistant to one drug, three to two drugs and one to three drugs. The number of reported cases of drug resistance has remained relatively constant during recent years. See Table 2.

There is continued concern about the majority (51%) of cases which occur in the outstate area, where access to medical care is limited and sporadic, especially for those patients with no third-party insurance coverage. The Diagnostic Services Program was developed to meet this need.

Vaccine-Preventable Diseases - 1993

(continued from page 23)
Mothers should be given a Missouri Immunization Record Card before leaving the hospital, with the date of the first hepatitis dose noted. The baby can receive the second and third doses (at ages one month and six months) through their local health agency.

Maintaining high levels of immunization is crucial to preventing outbreaks of the above deadly diseases. High immunization levels are also vital to preventing cases of invasive *Haemophilus influenza* disease (see pages 3 and 17 of this issue for additional information on the occurrence of this disease in Missouri). However, currently, fewer than half of all two-year-olds in Missouri are age-appropriately immunized.

REFERENCE

1. Centers for Disease Control and Prevention. DTP vaccine best protection against pertussis. Immunization Action News, March 7, 1994.

Table 1. Reported Pertussis Cases by Age Group and DTP Immunization Status, Missouri, 1993

Age Group	No. of Immunizations							
	0	1	2	3	4	5	Unknown	Total
0–6 mos.	40	22	13	2	0	0	3	80
7–11 mos.	4	4	7	3	0	0	2	20
1–6 yrs.	5	2	3	9	4	1	6	30
7–19 yrs.	1	3	1	1	2	1	2	11
20+ yrs	0	0	0	0	0	1	2	3
Total	**50**	**31**	**24**	**15**	**6**	**3**	**15**	**144**

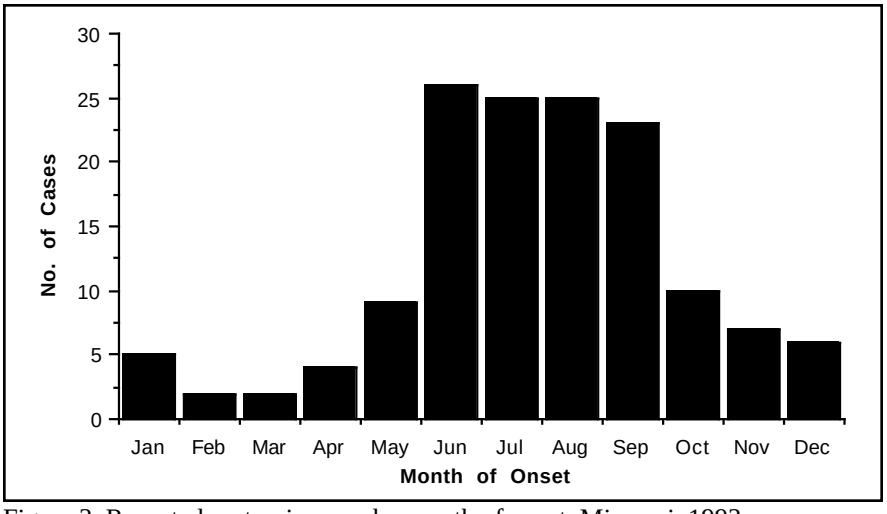


Figure 3. Reported pertussis cases by month of onset, Missouri, 1993.

State Public Health Laboratory - 1993 Annual Report

Metabolic Disease Screening

Infants screened	**78,351**
Presumptive positives:	
PKU	14
Hypothyroidism	328
Galactosemia	39
Sickle Cell	28
Other hemoglobinopathies	1,631

Microbiology

Enterics	**1,362**
Salmonella	486
Shigella	328
Campylobacter jejuni	23
E. coli O157:H7	37
Parasitology	**2,227**
Specimens with ova/parasites present	507
Entamoeba histolytica	15
Giardia lamblia	125
Ascaris lumbricoides	39
Hookworm	24
Trichuris trichura	24
Reference Bacteriology	**3,160**
Francisella tularensis	5
Haemophilus influenzae	62
Neisseria meningitidis	50
Bordetella pertussis	141
DNA Probe for Chlamydia/Gonorrhoeae	**42,201**
N. gonorrhoeae	616
C. trachomatis	2,760

Serology/Virology

HIV Serology	**85,439**
HIV antibody positive	1,051
Syphilis Serology	**16,703**
Sero-confirmed serologic reactive	1,477
Spinal fluid - reactive	4
Hepatitis A Serology	**1,529**
Positive	251
Hepatitis B Serology	**6,742**
Acute cases	3
Infectious patients	204
Not infectious but exposed	952
Measles, Mumps and Rubella (Diagnostic Serologies)	**104**
Measles (IgM positive)	4
Mumps (IgG two-fold rise in titer)	1
Prenatal rubella screens	7,273
Nonreactive patients	718
Viral Isolation	**1,695**
Influenza	431
Enterovirus	180
Herpes	1,048
Rabies	**2,145**
Positive specimens	35

Environmental Testing

Chemistry	
Total samples	**5,222**
Number of analyses	13,345
Bacteriology	
Water	
Private samples	**5,587**
Coliform positive	1,710
Fecal coliform positive	228
Public water supply samples	**28,975**
Coliform positive	890
Fecal coliform positive	109
Food/Dairy	**3,192**
Significant findings: *Staph. aureus* (Coagulase positive)	6
Excessive bacterial growth, yeast and mold (processed food, beverage samples)	106

State Public Health Laboratory Report

Newborn Screening — Hypothyroidism, Phenylketonuria,Galactosemia and Hemoglobinopathies

James Baumgartner, BS, MBA, Chief, Metabolic Disease Unit

	Jan 94	Feb 94	Total YTD
Specimens Tested	9,614	9,054	18,668
Initial (percent)	64.6%	65.0%	12,104
Repeat (percent)	35.4%	35.0%	6,564
Specimens: Unsatisfactory	110	108	218
HT Borderline	792	717	1,509
HT Presumptive	34	40	74
PKU Borderline	5	3	8
PKU Presumptive Positive	0	0	0
GAL Borderline	35	38	73
GAL Presumptive Positive	5	3	8
FAS (Sickle cell trait)	95	88	183
FAC (Hb C trait)	23	26	49
FAX (Hb variant)	9	11	20
FS (Sickle cell disease)	3	2	5
FSC (Sickle C disease)	1	0	1
FC (Hb C disease)	0	0	0

HT = Hypothyroidism, PKU = Phenylketonuria, GAL = Galactosemia, Hb = Hemoglobin, YTD = Year to Date

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The Managing Editor is H. Denny Donnell, Jr., MD, MPH, State Epidemiologist, assisted by Bill Schmidt, MPH, Director of the Division of Environmental Health and Epidemiology. Diane C. Rackers is the Production Manager. Questions or comments should be directed to (314) 751-6128 or toll free (800) 392-0272.

Alternate forms of this publication for persons with disabilities may be obtained by contacting the Missouri Department of Health, Division of Environmental Health and Epidemiology, P.O. Box 570, Jefferson City, MO 65102, (314) 751-6128. TDD users can access the preceding phone number by calling (800) 735-2966.

This newsletter can be recycled.

Second MMR is Not a Booster

Epidemics of measles involving large numbers of pre-schoolers, high school and college-aged students led, in 1989, to a recommendation for universal immunization with two doses of measles/mumps/rubella (MMR) vaccine. Data show that measles vaccine is effective in 95 percent of recipients. The second dose of measles vaccine is given, **not** as a booster for waning immunity, but rather to effectively immunize those five percent (1-in-20 individuals) who did not receive immunity with the initial dose.

The Advisory Committee on Immunization Practices (ACIP) recommends that the initial dose of MMR be routinely given at age 12–15 months of age and a second dose at school entry. Currently the State of Missouri requires that a child who entered kindergarten (or was five years old) as of the 1990–91 school year, is required to have two doses of measles-containing vaccine, at least 30 days apart, with the first dose on or after the first birthday, before he or she can attend school.

In the spring of 1994, Missouri had the largest outbreak of measles in the United States since 1992, with 156 cases reported. To date in 1994, 161 cases of measles have been reported statewide. All reported cases occurred in unimmunized or inadequately immunized individuals.

Missouri EPIDEMIOLOGIST

Volume XVI, Number 4 *August–October 1994*

The Vaccines for Children Program Continues

Paula Rosenberg
Bureau of Immunization

The Vaccines for Children (VFC) Program is alive and well, thanks to the efforts of the public and private medical community. Members of the private medical community, hospitals, federally funded health centers and rural health centers are being invited to partner with the Missouri Department of Health in the national Vaccines for Children Program.

The VFC program is a new federally funded and state-operated supply program that began October 1, 1994 for public providers and will begin in 1995 for private providers. The program is intended to help raise childhood immunization levels in the United States, especially among infants and young children.

The program will supply—at no cost to private health-care providers who agree to participate—up to $270 worth of federally purchased vaccine to be administered to each child in certain groups. Approximately 60 percent of Missouri's children may be expected to benefit from the VFC program.

A Delay

The United States Congress recently cancelled plans for a National Vaccine Distribution Center. This center was supposed to distribute the vaccine directly to the private sector participants. Plans for delivering the vaccine to our private sector partners will now have to be reworked. The anticipated delay should be no more than a few months.

Providers are encouraged to enroll now so that they can receive VFC vaccine as soon as it becomes available. Six weeks notice will be given so that providers may reduce their stock in anticipation of VFC vaccine.

Why Do We Need VFC?

Immunization rates in Missouri are too low. Only 52 percent of Missouri's two-year-olds, in both the public and private sector, have received all the immunizations they need in a timely fashion.

Today, the challenge to vaccinate children is tougher: vaccines cost more, new vaccines have been added to the schedule and access to immunizations for children is limited.

In Missouri, parents who could not afford the cost of a full series of immunizations for their children were referred by private physicians to public health clinics. As a result, patient loads at public health clinics have increased beyond capacity and the child's care has become splintered. This means that infants are not being immunized on time.

Who Is Eligible For VFC?

Those eligible for VFC include children from birth through 18 years of age who are in the following groups:

- No health insurance
- Native Americans/Alaska Natives
- Medicaid-enrolled.

Federally qualified health centers and rural health centers can also see any child who has health insurance that does not cover immunizations.

All other private providers may use publicly purchased vaccine to cover these underinsured children if the child's family income is 200 percent of poverty level or less.

(continued on page 2)

Inside this Issue...

(continued from page 1)
The local health agencies will continue to see any child who presents for immunizations, regardless of insurance or income level.

Verification that a child belongs to one of these groups is not required. One form, to be filled out on the first visit, is all that is required to enroll a child in this program.

What Do Providers Need To Do In Order To Participate?

Great effort has gone into this program to keep it as hassle-free as possible for busy providers. To participate in the VFC program, providers need to agree to:

1. Screen the patient for eligibility on the first visit;

2. Follow the immunization schedule established by the ACIP and Missouri state law;

3. Not charge for the VFC-supplied vaccine (although an administration fee may be charged);

4. Provide vaccine information materials as prescribed by law (required of all providers, regardless of their enrollment status in the VFC program).

A one-time provider enrollment form agreeing to these standards will be kept on file at the state health department.

Once providers receive their VFC vaccine, they need not worry about separating VFC vaccine from their other stock.

Providers will be welcome to enroll at any time during the program. However, those who wish to receive vaccine as soon as it becomes available should send in their enrollment forms as soon as possible.

The VFC program is only one part of the overall solution to increase immunization rates in Missouri. Parent and provider education, as well as other infrastructure improvements, will have to continue if we are to truly have an impact. However, this program should allow providers to use every opportunity to provide immunizations to our children.

Packets with more information on VFC, along with enrollment forms were sent out in August. If you or your facility have not received this packet, or if you would like more information about VFC, please call the **Bureau of Immunization at 1 (800) 219-3224.**

Laboratory Screening for Antibiotic-Resistant *Streptococcus pneumoniae*

Mahree Fuller Skala, M.A.
Bureau of Communicable Disease Control

The Council of State and Territorial Epidemiologists strongly encourages microbiology laboratories to routinely screen all pneumococcal isolates for penicillin resistance using a 1 μg oxacillin disc and to determine minimal inhibitory concentrations (for penicillin and other drugs likely to be used in treating invasive disease) for isolates found to be resistant on screening according to National Committee for Clinical Laboratory Standards guidelines.

Reports of antibiotic-resistant *S. pneumoniae* infection, including strains resistant to penicillin, extended spectrum cephalosporins and chloramphenicol, have been increasing since the 1980's. Limited active surveillance data from the Centers for Disease Control and Prevention (CDC) also documents an increase in antibiotic-resistant isolates since 1987. Recent surveys show that in some communities the problem is quite serious and that a substantial proportion of infections are due to highly and multi-resistant strains, while in other communities the proportions are low.

S. pneumoniae is the most frequent bacterial pneumonia in persons of all ages, the most common cause of bacteremia and acute otitis media in children and a leading cause of meningitis in the United States. There are an estimated 500,000 cases of pneumococcal pneumonia yearly in the United States. The overall annual incidence rate for pneumococcal bacteremia is 15–19/100,000 population. Young children and the elderly are at greatest risk for pneumococcal infections and complications, and the annual incidence rate of bacteremia is 100–160/100,000 population in children under two years of age and 50–70/100,000 population in adults over 65 years of age. Nonwhite persons, especially blacks, and HIV-infected persons are also at increased risk for pneumococcal bacteremia.

While the prevalence of antibiotic-resistant *S. pneumoniae* infection appears to be increasing, the magnitude and geographic distribution of the problem are not known. Prior to the emergence of antibiotic-resistant *S. pneumoniae*, most pneumococcal infections were treated empirically with penicillin or other beta-lactam drugs. Surveillance data are now needed to identify areas with high rates of antibiotic-resistant pneumococci to allow clinicians to better select empiric antimicrobial drug therapy for pneumococcal infections and allow public health officials to monitor national trends in resistance patterns. Such information may be useful for targeting selected areas for vaccine promotion campaigns.

CDC is in the process of establishing two demonstration Emerging Infections Programs in the United States, to study this and other emerging infectious disease threats to public health.

Reprinted in part from a position statement approved by the Council of State and Territorial Epidemiologists at their annual meeting in May, 1994.

Jasper County Missouri Superfund Site Blood-Lead and Urine-Cadmium Exposure Study

Bureau of Environmental Epidemiology

Introduction

Lead's poisonous effect on humans, especially children, has been well proven and documented by science. Consistent findings from several recent extensive studies indicate that lead causes harmful health effects on the development of unborn babies and young children, including abnormal nervous system and physical growth and lowered I.Q. scores. These harmful effects were associated with increased levels of lead in blood as low as ten micrograms lead per deciliter of blood (μg/dL).

The Centers for Disease Control and Prevention (CDC) has described childhood lead poisoning as one of the most common preventable environment-related health problems for children in the United States today. Enough is known about the sources and pathways of lead exposure for the CDC to establish a national goal to stop the problem of lead poisoning by the year 2012. Sources for lead exposure include air, food, water, dust and soil. Throughout human history, lead has been used in paints, glazes, eating utensils, plumbing, drugs and gasoline. In addition, Missouri citizens have been exposed to lead through mining, milling and smelting of lead ore. Missouri is the number one lead producing state in the nation.

The Jasper County Missouri Superfund Site Blood-Lead and Urine-Cadmium Exposure Study was conducted to determine if residents living in the Jasper County Superfund Site area (study population), an old lead mining, milling and smelting area in southwest Missouri, have blood-lead and urine-cadmium levels higher than residents living in nearby non-mining areas (control population). The health study's objectives, methods, results, conclusions and recommendations are outlined in this article.

Study Objectives

- To measure the amounts of lead in blood and cadmium in urine in a study population (people living in mining areas) and compare them with levels found in a control population (people living in a non-mining area).

- To determine the level of lead and cadmium contamination in soil, interior house paint, interior house dust and water in a study area and compare these with levels of contamination observed in a control area.

- To determine how a wide variety of blood and urine test results from the study and control groups compare to each other and to standard reference levels for the same tests.

- To determine the extent to which a wide variety of factors (demographic, environmental, behavioral, occupational and socio-economic) influence the differences of blood-lead and urine-cadmium levels in study and control populations.

Site History

The Jasper County Superfund Site is a portion of the old Tri-State Mining District. From 1850 to 1957, it was one of the largest lead-zinc mining areas in the world. Discovery of ore deposits in the area led to the building of mining camps during the pre-Civil War period on Turkey Creek and Center Creek in Jasper County, and at Spurgeon, Moseley and Granby in Newton County. From the 1840's through the Civil War, more than 200 widely scattered primitive log smelting furnaces operated in this area. These smelting furnaces were replaced with centrally located Scotch-hearth smelters located at Granby, Moseley and Center Creek. Mine production in the Missouri portion reached its peak in 1916, when more than 123 million rock-tons were processed to yield about 304 thousand tons of zinc concentrates and 41 thousand tons of lead concentrates.

The Missouri portion of the Tri-State Mining District has a north-south length of about 30 miles, from Granby north to Neck City, with mine tailings spread over approximately 240 square miles. Communities in the area include Joplin, Carterville, Carthage, Diamond, Duenweg, Granby, Oronogo, Ritchey and Webb City.

About eight million cubic yards of waste milling and mining tailings are scattered throughout the area. Some areas have been redeveloped for residential and industrial uses by leveling and mixing the remaining tailings with the soil. Open mine shafts, sunken areas having steep, unstable slopes and open pits with deep pools of water exist throughout the region. The general site is primarily uncontrolled and is routinely used for recreational purposes (i.e. fishing, hunting, scuba diving, swimming, ATV usage, etc.). In addition, water-quality problems result from underground flow of mine waters from open shafts, rainwater runoff and seepage from tailings piles and settling ponds. Chats (gravel or stone-sized pieces) have been used as railroad ballast, road materials and mixtures in asphalt paving and concrete. Sands and smaller sizes have been used for children's sandboxes and play areas and as abrasives, roofing granules, pipe coatings and sand for filter systems.

In the mid- and late 1980's, the Environmental Protection Agency (EPA) and the Missouri Department of Health (DOH) began soil sampling throughout the mine tailings area. Elevated levels of lead and cadmium were found in tailings materials believed to most likely cause high population exposure. In 1991, the DOH began a large-scale health study to determine how local residents had been and were being affected by the contaminated mine tailings.

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Study Methods

Biological Testing

Through a local population census, eligible persons were randomly selected for participation in the biological study phase. To be eligible for this phase, candidates had to be between 6 months and 45 years of age and residing at their current address for 60 days or more. The study population was divided into age groups: 6–71 months (children), 6–14 years (youth), and 15–44 years (adults). Participants in the blood and urine testing gave blood and urine samples that were then put through a variety of tests. Approximately 14 percent of the exposed children had blood-lead levels higher than or equal to ten micrograms of lead per deciliter of blood, while no children from the control group had elevated blood-lead levels.

To ensure quality control and assurance of the biomedical testing, contracts were made with Midwest Research Institute, Kansas City, Missouri, and with Environmental Health Laboratory Sciences, part of the CDC, to conduct the laboratory analyses.

Environmental Testing

Environmental sampling was conducted for lead and cadmium in drinking water, soil and house dust. After further census and house selection was conducted, a random sample group of 125 study households (including elevated blood-lead homes) and 26 control households was finalized. Because of available resources, EPA conducted the sampling under an agreement with the Agency for Toxic Subtances and Disease Registry (ATSDR) and DOH.

Study Results

The biological results shown in Table 1 and the environmental results shown in Table 2 are not intended to reflect the level of risk for lead exposure or lead poisoning for area residents. The results given are to show only the difference in blood-lead and urine-cadmium levels between the study and control groups, and to show the difference in the levels of lead and cadmium in various environmental media between the two areas. It is important to remember that these numbers reflect averages among the tested age groups and media and do not show individual test results.

Table 1. Average Blood-Lead and Urine-Cadmium Levels by Group

Age Group	Study Group	Control Group
6–71 months		
Blood-Lead (µg/dL)	6.25	3.59
Urine-Cadmium (µg/dL)	0.07	0.08
6–14 years		
Blood-Lead (µg/dL)	3.61	2.46
Urine-Cadmium (µg/dL)	0.114	0.087
15–44 years		
Blood-Lead (µg/dL)	3.44	2.22
Urine-Cadmium (µg/dL)	0.419	0.309

Table 2. Average Lead and Cadmium Levels by Material Tested and Group

Material Tested	Study Group	Control Group
Lead in Dust (µg/g)	608.00	209.000
Lead in Water (µg/L)	2.62	2.120
Lead in Paint (µg/cm^2)	1.38	0.412
Lead in Soil (mg/kg)	599.00	91.100
Cadmium in Dust (µg/g)	8.60	4.850
Cadmium in Water (µg/L)	1.51	0.225
Cadmium in Soil (µg/kg)	11.10	2.590

Study Conclusions

The Jasper County blood-lead and urine-cadmium study evaluated one exposed population and one control population for exposure to lead and cadmium mining wastes. It evaluated four specific objectives and found:

- The blood-lead levels were much higher in the study group, compared to the control group. The difference in urine-cadmium levels between the two groups was not significant.
- Levels of lead and cadmium in various environmental media (soil, dust, interior paint) were much higher in the study area, compared to the control area.
- There were very few consistent statistically significant differences in the blood and urine tests between the study and control groups.
- Environmental exposure to soil was the most important factor in the differences in blood-lead levels between the study and control groups.

The DOH contacted each participant in the health study and notified them of their own blood and urine test results. The study also showed that environmental, behavioral and socio-economic factors had the greatest influence on the blood-lead levels between the study and control groups. Other factors such as age, race and occupation did not have significant influence on blood-lead levels in the study and control groups.

The CDC suggests that all children be tested for blood-lead content, no matter where they live. Further, parents in the study area should be aware of the greater risk of lead poisoning posed to their children because of increased lead lev-

els in local soil and dust. Because of these increased levels, parents in the Jasper County area should have their children's blood tested for lead. Parents should also keep in mind that personal and household cleanliness is a key to keeping lead exposure to a minimum. Children should also be kept from playing in non-grassed soil and dust.

Parents and other local residents should know that the lead levels found in the Jasper County area do not present an immediate health risk to children or adults. However, study results do show that if children (age 6 months to 6 years) continue to be exposed to increased levels of lead in soil and dust, they are at risk of damage to their developing nervous systems. This damage could cause the child to show I.Q. scores four to five points lower than unexposed children their own age. While this does not have serious physical implications, it could have a negative impact on the child's potential to achieve in school and the workplace.

Study Recommendations

- Study results found increased blood-lead levels due to exposure to contaminated soils in the Jasper County Superfund Site. The DOH, therefore, recommends that exposure to the lead-contaminated soil in the study area be reduced.
- Harmful effects to the nervous system of unborn babies and young children are considered to be the health problems of concern due to blood-lead poisoning. The study did not attempt to evaluate nervous system effects, such as I.Q. scores from standard tests given in local school systems. The DOH recommends a study be conducted to evaluate the harm lead can cause to the nervous system, based on school records and standard test scores.
- For future studies of this kind, the DOH recommends environmental soil and dust samples for lead be examined to determine the percent of the contribution from various sources of environmental lead.

State Public Health Laboratory Report

Newborn Screening — Hypothyroidism, Phenylketonuria, Galactosemia and Hemoglobinopathies

James Baumgartner, B.S., M.B.A., Chief, Metabolic Disease Unit

	Mar 94	Apr 94	Total YTD
Specimens Tested	10,724	9,282	38,674
Initial (percent)	64.5%	63.6%	24,927
Repeat (percent)	35.5%	36.4%	13,747
Specimens: Unsatisfactory	126	127	471
HT Borderline	803	651	2,963
HT Presumptive	43	24	141
PKU Borderline	10	4	22
PKU Presumptive Positive	1	1	2
GAL Borderline	36	43	152
GAL Presumptive Positive	3	3	14
FAS (Sickle cell trait)	97	86	366
FAC (Hb C trait)	39	21	109
FAX (Hb variant)	13	11	44
FS (Sickle cell disease)	2	1	8
FSC (Sickle C disease)	0	0	1
FC (Hb C disease)	0	0	0

	May 94	Jun 94	Total YTD
Specimens Tested	9,360	10,587	58,621
Initial (percent)	63.7%	65.8%	37,853
Repeat (percent)	36.3%	34.2%	20,768
Specimens: Unsatisfactory	105	100	676
HT Borderline	565	863	4,391
HT Presumptive	35	40	216
PKU Borderline	29	9	60
PKU Presumptive Positive	0	0	2
GAL Borderline	77	192	421
GAL Presumptive Positive	4	3	21
FAS (Sickle cell trait)	80	98	544
FAC (Hb C trait)	20	27	156
FAX (Hb variant)	13	14	71
FS (Sickle cell disease)	1	3	12
FSC (Sickle C disease)	1	1	3
FC (Hb C disease)	2	0	2

HT = Hypothyroidism, PKU = Phenylketonuria, GAL = Galactosemia, Hb = Hemoglobin, YTD = Year to Date

School Immunization Requirements Amended for 1994–95 School Year

Mary Ann Harder
Bureau of Immunization

The Missouri Department of Health rule concerning immunization requirements for school attendance (19 CSR 20-28.010) was amended in 1994. These changes became effective with the beginning of the 1994–95 school year. The three major areas affected include: vaccine requirements, the grace period and the religious exemption.

Vaccine Requirement Changes

Students who enrolled in kindergarten or the first grade during or after the 1990–91 school year (kindergarten through fourth grade) are now required to meet the following requirements:

- Three doses of DTP with the last dose on or after their fourth birthday, **and**
- Three doses of OPV with the last dose on or after their fourth birthday

All other immunization standards remain the same.

No Grace Period

The 15-day grace period has been removed. All students must present proof of compliance with the immunization requirements for school attendance on the first day of school. This applies to all students, including transfer students. The only exception will be for homeless children, who will be granted 24 hours to provide proof of immunization, or to show that they are in the process of completing the immunizations.

Change in Religious Exemption

The law still requires documentation of all religious exemptions to immunizations. However, parents are no longer required to identify their religion when completing the required Religious Immunization Exemption form.

Additional information regarding immunization requirements for school attendance may be obtained from the district immunization representative located in each of the district health offices, or by contacting the **Bureau of Immunization at (314) 751-6133.**

Summary Chart of the Missouri School Immunization Rule*

Vaccine	No. of Doses	Type	Timing
Measles	2§	Usually MMR (Measles/Mumps/Rubella)	Two doses on or after first birthday, separated by at least 30 days, for students who started kindergarten,or who were 5 or 6 years of age, as of and after the 1990-91 school year.
Rubella	1	Usually MMR (Measles/Mumps/Rubella)	On or after first birthday.
Mumps	1	Usually MMR (Measles/Mumps/Rubella)	On or after first birthday.
Polio¶	At least 3	Usually TOPV (Trivalent Oral Polio)	Last dose must have been at age 4 or older.¥
Diphtheria†	At least 3 (+10 year booster)	DTP, DT, Td	Last dose must have been at age 4 or older.¥
Tetanus†	At least 3 (+10 year booster)	DTP, DT, Td	Last dose must have been at age 4 or older.¥
Pertussis†	At least 3	DTP	Applies to students 6 years of age and younger. Last dose must have been at age 4 or older.

* These are the minimum requirements for school attendance in Missouri. The Department of Health Immunization Schedule reflects the optimal immunization recommendations of the Advisory Committee on Immunization Practices and the American Academy of Pediatrics.
§ One dose of live measles vaccine on or after the first birthday is required for all students who started kindergarten prior to the 1990-91 school year.
¶ No more than 4 doses of polio are required for attendance.
¥ Applies to those students who started kindergarten, or who were 5 or 6 years of age, as of and after the 1990-91 school year.
† No more than 6 doses of DTP should be given to students less than 7 years of age.

Recommendations of the U.S. Public Health Service Task Force on the Use of Zidovudine to Reduce Perinatal Transmission of Human Immunodeficiency

The following is an excerpt from the Recommendations of the U.S. Public Health Service Task Force on the Use of Zidovudine to Reduce Perinatal Transmission of Human Immunodeficiency Virus, published in the August 5, 1994, MMWR Recommendations and Reports (Vol. 43, No. RR-11). Providers should consult the complete article for a fuller discussion of the role of AZT in reducing the transmission of HIV from an infected mother to her infant.

These recommendations update the interim guidelines[1] developed by the U.S. Public Health Service for the use of zidovudine (ZDV) to reduce the risk for perinatal transmission of human immunodeficiency virus (HIV) infection. The recently reported results of AIDS Clinical Trials Group (ACTG) Protocol 076 demonstrated that ZDV administered to a selected group of HIV-infected pregnant women and their infants can reduce the risk for perinatal HIV transmission by approximately two-thirds[1,2]. The regimen used in this trial included antenatal oral administration of ZDV beginning at 14–34 weeks of gestation and continuing throughout pregnancy, followed by intrapartum intravenous ZDV and postnatal oral administration of ZDV to the infant for 6 weeks after delivery. See Table 1. This use of ZDV has the potential to substantially reduce the rate of perinatal transmission, which would reduce overall child mortality. However, the results of this study are directly applicable only to HIV-infected women with characteristics similar to those of the women who entered the study, and the long-term risks of ZDV used in this manner are not known. See Table 2.

Table 1. Zidovudine regimen from AIDS Clinical Trials Group Protocol 076

- Oral administration of 100 mg of zidovudine (ZDV) 5 times daily, initiated at 14–34 weeks of gestation and continued throughout the pregnancy.
- During labor, intravenous administration of ZDV in a 1-hour loading dose of 2 mg per kg of body weight, followed by a continuous infusion of 1 mg per kg of body weight per hour until delivery.
- Oral administration of ZDV to the newborn (ZDV syrup at 2 mg per kg of body weight per dose every 6 hours) for the first 6 weeks of life, beginning 8–12 hours after birth.

Table 2. Eligibility criteria for HIV-infected pregnant women participating in AIDS Clinical Trials Group Protocol 076

- Pregnancy at 14–34 weeks of gestation.
- No antiretroviral therapy during the current pregnancy.
- No clinical indications for antenatal antiretroviral therapy.
- CD4+ T-lymphocyte count ≥ 200 cells/μL at the time of entry into the study.

Enrolled women were assigned randomly to receive a regimen of either ZDV or placebo. The estimated transmission rate was 25.5 percent among the 184 children in the placebo group (95 percent confidence interval [CI]=18.4–32.5%), compared with 8.3 percent among the 180 children in the ZDV group (95% CI=3.9–12.8%). The difference in the estimated transmission rate between the two groups was statistically significant (p=0.00006). ZDV treatment did not appear to delay the diagnosis of HIV infection.

Observed toxicity specifically attributable to ZDV was minimal among the women in this study. Adverse effects such as anemia, neutropenia, thrombocytopenia, and liver chemistry abnormalities were reported as frequently among women receiving placebo as among women receiving ZDV. Six women—three in each treatment group—discontinued therapy because of toxicity attributed to the study drug.

Serial sonographic evaluations for fetal growth and amniotic fluid volume as conducted in the study (at entry and every four weeks from 28 weeks of gestation until delivery) demonstrated no differences between pregnancies in women who had received placebo or ZDV. Birth parameters (gestational age; birth weight, length and head circumference; and Apgar scores) were similar among infants born to women in either group. The occurrence of major or minor congenital abnormalities was approximately equal between the two groups, and no pattern in the type of abnormalities was observed.

The infants in the study tolerated the ZDV therapy well. The only adverse effect observed more frequently among infants in the ZDV treatment group was mild, transient anemia.

This clinical trial demonstrated that the ACTG Protocol 076 ZDV regimen can substantially reduce perinatal HIV trans-

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mission. However, several important limitations should be noted:

- Perinatal HIV transmission was still observed despite drug therapy.
- The efficacy of this therapy is unknown for HIV-infected pregnant women who have advanced disease, who have received prior antiretroviral therapy, or who have ZDV-resistant virus strains.
- Although the ZDV regimen used in this trial was not associated with serious short-term adverse effects, such effects may be observed when this use of ZDV becomes more widespread.
- The long-term risks for the child associated with exposure to ZDV in utero and early infancy have not been determined.
- It is not known if use of ZDV during pregnancy will affect the drug's efficacy for the woman when it becomes clinically indicated for her own health.

General Principles Regarding Treatment Recommendations

The following treatment recommendations have been formulated to provide a basis for discussion between the woman and her health-care provider about the use of ZDV to reduce perinatal transmission. HIV-infected women should be informed of the substantial benefit and short-term safety of ZDV administered during pregnancy and the neonatal period observed in ACTG Protocol 076. However, they also must be informed that the long-term risks of ZDV therapy to themselves and their children are unknown. A woman's decision to use ZDV to reduce the risk for HIV transmission to her infant should be based on a balance of the benefits and potential risks of the regimen to herself and to her child.

Discussion of treatment options should be noncoercive, and the final decision to accept or reject ZDV treatment recommended for herself and her child is the right and responsibility of the woman.

Table 3 lists different situations involving HIV-infected pregnant women which may be encountered in clinical practice, along with recommendations for ZDV use. All potential clinical situations cannot be enumerated, and, in many cases, definitive evidence upon which to base a recommendation is not currently available. Therefore, each pregnant woman and her health-care provider must consider the potential benefits, unknown long-term effects, and gaps in knowledge relating to her clinical situation. Health-caregivers and institutions should provide culturally, linguistically and educationally appropriate information and counseling to the HIV-infected woman so that she can make informed decisions.

Recommendations for Monitoring the ZDV Regimen for Mothers and Infants

Maternal Monitoring

HIV-infected pregnant women should be monitored in accordance with previously published guidelines[3,4]. Monitoring during pregnancy should include monthly assessment for ZDV-associated hematologic and liver chemistry abnormalities. Indications of toxicity that might require interrupting or stopping the dose of ZDV include:

a) hemoglobin <8 gm/dL,

b) absolute neutrophil count <750 cells/μL, **or**

c) AST (SGOT) or ALT (SGPT) greater than five times the upper limit of normal.

CD4+ T-lymphocyte counts should be monitored to determine if prophylaxis for opportunistic infections, such as *Pneumocystis carinii* pneumonia (PCP), should be initiated. Pregnant HIV-infected women with CD4+ T-lymphocyte counts <200 cells/μL should receive appropriate PCP prophylaxis. If the CD4+ T-lymphocyte count is <600 cells/μL, the evaluation should be repeated each trimester. CD4+ T-lymphocyte counts should be measured at six weeks and six months postpartum to evaluate if antiretroviral therapy is indicated.

Fetal Monitoring

Antepartum testing, including sonographic and nonstress testing and intrapartum fetal monitoring, should be performed only as clinically indicated, not specifically because the patient is being treated with ZDV during pregnancy.

Infant Monitoring

A complete blood count and differential should be performed at birth as a baseline evaluation. Repeat measurements of hemoglobin are recommended at 6 and 12 weeks of age. ZDV should be administered with caution to infants born with severe anemia (hemoglobin <8 gm/dL), and treatment of the anemia and intensive monitoring are warranted if the drug is administered.

REFERENCES

1. CDC. Zidovudine for the prevention of HIV transmission from mother to infant. MMWR 1994;43:285-7.
2. National Institute of Allergy and Infectious Diseases. Clinical alert: important therapeutic information on the benefit of zidovudine for the prevention of the transmission of HIV from mother to infant. Bethesda, MD: National Institutes of Health, National Institute of Allergy and Infectious Diseases, February 20, 1994.
3. Sperling RS, Stratton P, Obstetric Gynecologic Working Group of the AIDS Clinical Trials Group of the National Institute of Allergy and Infectious Diseases. Treatment options for human immunodeficiency virus-infected pregnant women. Obstet Gynecol 1992;79:443-8.
4. El-Sadr W, Oleske JM, Agins BD, et al. Evaluation and management of early HIV infection. Clinical practice guideline no. 7. AHCPR publication no. 94-0572. Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, US Department of Health and Human Services, January 1994.

Table 3. Summary: Clinical Situations and Recommendations for Use of Zidovudine* to Reduce Perinatal HIV Transmission

I. Pregnant HIV-infected women with CD4+ T-lymphocyte counts ≥ 200/μL who are at 14–34 weeks of gestation and who have no clinical indications for ZDV and no history of extensive (>6 months) prior antiretroviral therapy.

Recommendation: The health-care provider should recommend the full ACTG Protocol 076 regimen to all HIV-infected pregnant women in this category. This recommendation should be presented to the pregnant woman in the context of a risk-benefit discussion: a reduced risk of transmission can be expected, but the long-term adverse consequences of the regimen are not known. The decision about this regimen should be made by the woman after discussion with her health-care provider.

II. Pregnant HIV-infected women who are at >34 weeks of gestation, who have no history of extensive (>6 months) prior antiretroviral therapy and who do not require ZDV for their own health.

Recommendation: The health-care provider should recommend the full ACTG Protocol 076 regimen in the context of a risk-benefit discussion with the pregnant woman. The woman should be informed that ZDV therapy may be less effective than that observed in ACTG Protocol 076, because the regimen is being initiated late in the third trimester.

III. Pregnant HIV-infected women with CD4+ T-lymphocyte counts <200/μL who are at 14–34 weeks of gestation, who have no other clinical indications for ZDV, and who have no history of extensive (>6 months) prior antiretroviral therapy.

Recommendation: The health-care provider should recommend initiation of antenatal ZDV therapy to the woman for her own health benefit. The intrapartum and neonatal components of the ACTG Protocol 076 regimen should be recommended until further information becomes available. This recommendation should be presented in the context of a risk-benefit discussion with the pregnant woman.

IV. Pregnant HIV-infected women who have a history of extensive (>6 months) ZDV therapy and/or other antiretroviral therapy before pregnancy.

Recommendation: Because data are insufficient to extrapolate the potential efficacy of the ACTG Protocol 076 regimen for this population of women, the health-care provider should consider recommending the ACTG Protocol 076 regimen on a case-by-case basis after a discussion of the risks and benefits with the pregnant woman. Issues to be discussed include her clinical and immunologic stability on ZDV therapy, the likelihood she is infected with a ZDV-resistant HIV strain, and, if relevant, the reasons for her current use of an alternative antiretroviral agent (e.g., lack of response to or intolerance of ZDV therapy). Consultation with experts in HIV infection may be warranted. The health-care provider should make the ACTG Protocol 076 regimen available to the woman, although its effectiveness may vary depending on her clinical status.

V. Pregnant HIV-infected women who have not received antepartum antiretroviral therapy and who are in labor.

Recommendation: For women with HIV infection who are in labor and who have not received the antepartum component of the ACTG Protocol 076 regimen (either because of lack of prenatal care or because they did not wish to receive antepartum therapy), the health-care provider should discuss the benefits and potential risks of the intrapartum and neonatal components of the ACTG Protocol 076 regimen and offer ZDV therapy when the clinical situation permits.

VI. Infants who are born to HIV-infected women who have received no intrapartum ZDV therapy.

Recommendation: If the clinical situation permits and if ZDV therapy can be initiated within 24 hours of birth, the health-care provider should offer the ACTG Protocol 076 postpartum component of 6 weeks of neonatal ZDV therapy for the infant in the context of a risk-benefit discussion with the mother. Data from animal prophylaxis studies indicate that, if ZDV is administered, therapy should be initiated as soon as possible (within hours) after delivery. If therapy cannot begin until the infant is >24 hours of age and the mother did not receive therapy during labor, no data support offering therapy to the infant.

*These recommendations do not represent approval by the Food and Drug Administration (FDA) or approved labeling for the particular product or indications in question.

A Media Illusion of an Epidemic

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FLESH-EATING VIRUS INVADES THE U.S. screamed the headline of the Weekly World News (28 June 1994) in supermarkets throughout the country. For weeks, the electronic and print media hammered away with what was perceived to be an epidemic of dire proportions. From an alleged epidemic in England the disease in question was seen to be spreading across the world. But was there any truth to the stories that bombarded the public?

The answer is yes and no. Yes, there were reports of invasive group A streptococcal infection (GAS) with necrotizing fasciitis in Gloucestershire, England. But no, it was not an epidemic (MMWR 43:401,1994). Yes, invasive GAS with necrotizing fasciitis was occurring in residents of this country and some cases were fatal. No, it is not a new disease and it is not epidemic.

GAS has been a significant threat as a human pathogen for centuries. Not only have there been numerous recurrent outbreaks of GAS infections in the past, but also both the suppurative and the nonsuppurative sequelae have been accompanied by considerable morbidity and mortality. And despite the liberal use of antibiotics, GAS infections remain common, e.g. pharyngitis, scarlet fever, and recent outbreaks of rheumatic fever (Curr Opinion Infec Dis 4:621, 1991). And, there is no doubt that in the past few decades, the epidemiology of GAS infections has been changing (Pediatr Infec Dis J 13:557,1994). Based on a four state prospective active surveillance program conducted between 1989–1991, the Centers for Disease Control and Prevention (CDC) estimated that 10,000–15,000 cases of invasive GAS occur annually in the United States, with 5–10% of patients developing necrotizing fasciitis. The case fatality rate for this subset of GAS cases is 28%, or approximately 140 – 420 deaths per year. Since 1991, there has been no active surveillance for invasive GAS in the United States or Missouri and the Kansas City Health Department maintains no statistics on invasive GAS in the community.

Necrotizing fasciitis is a severe infection that leads to necrosis of the subcutaneous tissue and adjacent fascia. The usual etiology is a mixture of aerobic and anaerobic organisms, but GAS alone may be responsible. This condition is a component of the case definition for GAS toxic-shock syndrome (JAMA 269:390,1993). The organisms reach the subcutaneous tissue by extension from a contiguous infection or trauma to the area, including surgery (Lancet 343: 1376,1994). There is widespread damage to surrounding tissue, and occlusion of small subcutaneous vessels leads to dermal gangrene. Characteristically, streptococcal gangrene begins at the site of trivial or inapparent trauma (Pediatr Infec Dis J 13:561,1994). Within 24 hours of the initial lesion, which frequently is only mild erythema, there is aggressive development of swelling, heat erythema and tenderness with rapid spreading proximally and distally from the original focus. During the next 24 – 48 hours the erythema darkens, changing from red to purple and then to blue, and blisters and bullae form that contain clear yellow fluid. On the 4th or 5th day, the purple areas become frankly gangrenous. From the 7th to 10th day the line of demarcation becomes sharply defined and the dead skin begins to separate at the margins or breaks in the center revealing extensive necrosis of the subcutaneous tissue. In more severe cases the process advances rapidly until several large areas of skin have become gangrenous and systemic toxicity renders the patient dull, mentally "cloudy," delirious or even unresponsive. Extensive surgical incision and debridement is the mainstay of treatment, with concomitant antibiotic therapy. There are various other necrotizing conditions that are clinically very difficult to distinguish from one another and from necrotizing fasciitis. So from an epidemiological viewpoint, do all of the reported cases in the media truly satisfy the case definition of invasive GAS with necrotizing fasciitis?

Microbiologically, *Streptococcus pyogenes* and Lancefield group A streptococcus are more or less synonymous, although the type-specific antigen A can be found in other streptococcal species. Group A streptococci can be further subtyped by serology on the basis of the T and M protein antigens within the bacterial cell wall. Most strains of *S. pyogenes* produce one or more of the

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Case Definition for GAS With Necrotizing Fasciitis

Confirmed Case: a patient presenting with necrosis of subcutaneous tissue together with severe systemic illness (including one or more of: sudden death; shock with a systolic blood pressure of less than 90 mm Hg; disseminated intravascular coagulation; system failure such as respiratory, hepatic or renal failure) and with group A streptococcus isolated from the affected site or from a normally sterile site.

Probable Case: a patient who fulfills the clinical definition above and who has the following serological or histological evidence of streptococcal infection, but without a culture of group A streptococcus from the affected site or from a normally sterile site. Serological evidence: a fourfold or greater rise in antistreptolysin O or antiDNAse B titers. Histological evidence: gram-positive cocci only in sections from affected tissue. (Lancet 343:1376,1994)

Fifth Disease: The Mild Mannered Rash Gains Importance

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For many years, fifth disease was viewed as an unimportant rash illness of children. More recently, studies have shown that the virus can be responsible for serious complications in certain individuals. The main concern in school health is the risk associated with pregnant women.

Fifth disease is a viral illness caused by the human parvovirus B19. The illness is classically characterized by mild systemic symptoms, usually non-febrile, and a distinctive rash. On the face, the rash is intensely red with a "slapped cheek" appearance. A lace-like rash can also be seen on the arms, trunk, buttocks and thighs. The rash can subside and recur with environmental changes, such as exposure to sunlight or heat, for weeks and sometimes even for months.

Infection with human parvovirus B19 presents in many ways, including asymptomatic infection, a mild respiratory infection with no rash, a rash that is atypical for this disease, arthritis in adults (with no other symptoms), chronic anemia in immunodeficient persons, and aplastic crisis in persons with chronic hemolytic anemia. In adults, the rash is often atypical or absent, but the arthritis or arthralgias may last days to months. It is estimated that 25 percent or more of B19 infections may be asymptomatic. Differentiation from rubella and scarlet fever is often necessary.

While anyone can become infected, the disease seems to occur more often in elementary school-age children as single cases or in outbreaks, which often occur during the spring. The virus is most likely spread by respiratory secretions. However, in outbreaks it is not known whether the spread occurs through direct personal contact, airborne droplets or contaminated objects. The virus may also be transmitted by blood products and from mother to fetus.

The incubation period is usually from 4 – 14 days, but may be as long as 20 days. Communicability is greatest before onset of rash in those persons with rash illness alone; these persons are probably not communicable after onset of rash. Persons with aplastic crisis are communicable up to one week after onset of symptoms. Immunosuppressed persons with chronic infection and severe anemia may be communicable for months to years. There is no specific treatment for parvovirus B19 and most patients require only supportive care. Chronic infections in immunodeficient persons may be treated with intravenous immunoglobulin therapy.

Clinical presentation and epidemiologic information usually provide the basis for diagnosis. Laboratory diagnosis can be made by assaying maternal serum for B19-specific IgM antibody, the presence of which confirms infection within the past several months. The presence of IgG antibody indicates prior infection and immunity. A recent study demonstrated the efficacy of polymerase chain reaction (PCR) for diagnosis of fetal parvovirus B19 infection. Because PCR is extremely sensitive (in fact, it may be too sensitive), results must be analyzed carefully for correlation with clinical infection.

Parvovirus B19 appears endemically and in outbreaks in school settings and carries a potential risk for pregnant adult contacts, including fetal hydrops and fetal death. Clinical awareness of active infection in the fetus is important because treatment of the fetus with intrauterine blood transfusions can be lifesaving. However, a recent study revealed that some cases of untreated fetal hydrops caused by this virus may resolve spontaneously and that intrauterine blood transfusions may have contributed to the death of one infant; thus clinicians must keep in mind that the disease at times is self-limited. Assessing the risk for teachers and day care workers who are exposed to children infected with parvovirus B19 is complicated because of the wide variation in outcome of infection with this virus. Because there is little or no virus present in respiratory secretions by the time the rash appears, there is no reason to isolate identified cases.

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Recommendations for School Nurses and Administrators

1. Report rash illnesses to your local health department. Since several rashes resemble each other and because there has been a recurrence of measles in Missouri care must be taken in identifying rash illnesses.
2. Children with B19 infection may attend school since they are no longer infectious after the rash appears.
3. Pregnant women working in schools with a high likelihood of exposure should be informed of the potential risk, even though it is relatively low.
4. Routine exclusion of pregnant women from the school setting where cases of fifth disease are occurring is not recommended. However, these women should be advised to consult with their physician, who may or may not advise testing for antibody status.
5. Individuals with chronic hemolytic anemia and immunodeficiencies are most likely to develop complications to B19 infection and thus should avoid exposure in outbreak settings.
6. Routine hygienic practices, including hand washing, covering the mouth and nose when sneezing or coughing and the proper disposal of tissues containing respiratory secretions, can lessen transmission of infection.

Heading Off the Problem of Pediculosis

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In recent years, infestation with *Pediculus humanus capitis* has assumed epidemic proportions in some regions of the United States, particularly among children 3 – 10 years of age. As can be seen in Table 1, there has been a decrease in reported cases of pediculosis in Missouri over the past five years. We are not sure why this decrease has occurred, but would like to believe it's a result of implementation of "no-nit" policies in schools and child care centers.

Table 1. Reported pediculosis cases by year, Missouri, 1989–93

Year	Cases Reported
1989	10,909
1990	12,047
1991	9,071
1992	8,635
1993	7,280

Pediculus humanus capitis is one of three species of lice which infest man. The other two species are *P. humanus corporis*, the body louse; and *Phthirus pubis*, the pubic, or crab, louse. The head louse is a bloodsucking insect found on the hair near the surface of the scalp, especially behind the ears and at the nape of the neck. Head lice are very small, approximately two to three millimeters long, and vary in color depending on the complexion of the human host; they appear darker on a host with dark skin and hair, and lighter on a host with light skin and hair. Their six legs, equipped with hook-like claws and opposing digits, enable them to grasp the hair shaft.

The lifespan of a head louse is believed to be about one month. During her lifetime, a female will deposit three to four eggs a day. Live eggs, called nits, are creamy yellow to gray color, darkening to tan or coffee color as they mature. They are oval in shape and firmly attach to the hair shaft close to the scalp by a cement-like substance. Eggs take about one week to hatch, and the emerging nymphs mature in eight to nine days.

Table 2. Pediculicides Available in the United States

Generic Name	Brand Name	Lice Killing Time	Application Time*	Ovicidal Activity
Permethrin	Nix	10–15 min.	10 min.	70–80%
Pyrethrins	A200, RID, R&C	10–23 min.	10 min.	70–80%
Lindane**	Kwell, Scabene, Kwildane, Gamene	140–230 min.	4 min.	45–70%

* Manufacturer recommended
** Prescription required

NOTE: Use of brand names is for identification purposes only and does not constitute endorsement by the DOH. There may be other brand names on the market which are not listed due to space constraints.

Both nymphs and adult lice feed on human blood. The skin is penetrated by the louse's mouth parts and saliva is poured in to prevent the blood from clotting. Itching, which is the major symptom of louse infestation, is caused by an inflammatory reaction to the saliva. Secondary infections may occur as a result of the scratching.

Transmission

Head lice are transmitted through direct contact with an infested person or indirectly via contact with infested garments (hats, caps, scarves), combs, bedding and such items as headphones and batting helmets. Studies suggest that person-to-person spread probably occurs more frequently than transmission by fomites. The reason is that head lice require the warm moist environment of the scalp and a frequent blood meal. Lice which leave the host will usually die within 6 to 20 hours.

Pediculosis is not indicative of uncleanliness and occurs in all socioeconomic groups. Race, however, is a significant factor with the prevalence of infestation being 35 times higher in whites and other races than in blacks.

Diagnosis

Diagnosis of pediculosis is made by observing lice or nits on the hair and scalp. A hair with a suspect nit attached should be removed and examined under a microscope or with a magnifying hand lens to confirm the diagnosis. Care should be taken to differentiate between viable nits and hair casts, dandruff or globules of hair spray, which will usually slide right off the hair. Nits, however, will stick to the hair shaft.

Magnification will reveal viable nits to be uniform in shape while other particles will be irregularly shaped. Empty nit casings have been described as white to dull yellow.

Treatment

Infested persons and all potential lice-carrying objects with which they have had contact should be treated. The National Pediculosis Association (NPA) recommends that persons with underlying health conditions, history of seizures or epilepsy, those on other medications, nursing or pregnant women, and children under 2 years of age who are in-

fested, contact their physician prior to using a pediculicide. There are several types of preparations on the market to treat head lice. See Table 2.

NOTE: Preparations containing lindane have been used since the early l950s, are still being used today and require a prescription. Although Kwell has been withdrawn from production, lindane is still being marketed under its generic name and other labels. Lindane is the most toxic and probably the least effective product available. Since 1983 the NPA has maintained that the potential toxicity of lindane outweighs any possible benefits it offers as a pediculicide and has worked diligently to alert the public to the potential adverse affects of lindane preparations. The Missouri Department of Health (DOH) does not advocate the use of lindane preparations.

There are several over-the-counter preparations on the market which will effectively eradicate head lice and are less toxic and more effective than lindane. A creme rinse preparation containing permethrin usually requires only one application, since residual activity on the hairs is effective in killing any lice that hatch from still viable eggs. Most shampoo preparations currently available (pyrethrins) do not have residual activity and a second application is required seven to ten days after the first. This is intended to eliminate lice that hatch after the first application of shampoo. Be sure to read and carefully follow package directions.

Whatever method is used, the DOH urges complete and thorough removal of nits with a specially designed nit comb. Nit removal is essential in preventing reinfestation and is recommended by the DOH even if product marketing information deems nit removal unnecessary.

Using hot water (130°F or 55°C) and detergent, machine wash all washable clothing and bed linens that have been in contact with the infested person. Drying clothing for 20 minutes at a high heat setting will also destroy nits. Dry cleaning will kill lice and nits. Combs and brushes should be soaked for one hour in a two percent Lysol solution or placed in hot water (150°F) for five to ten minutes. Be aware that some articles may be damaged by heat.

Family members should be inspected and treated only if they are infested. However, bedmates should be treated prophylactically. Bed linens and clothing which have been in contact with the infested person should be washed or dry cleaned. Home disinfestation may be accomplished by thoroughly vacuuming the mattress, carpets, upholstered furniture and car upholstery.

Fumigation or spraying of schools and homes is not recommended and is strongly discouraged by many health professionals, including those at the Centers for Disease Control and Prevention (CDC) and the NPA. Pediculicides are more toxic in their vaporous state and the potential health risk is unwarranted. There is no evidence available which indicates that use of environmental insecticides brings an outbreak of head lice under control faster than not using them.

Control

In order to stay ahead of the problem, it is wise to conduct several school-wide screenings during each school year, preferably during the first week of school, the week following Christmas vacation and again near the end of the school year.

When a case is reported in a school, close contacts such as classmates and friends should be examined for evidence of infestation. The DOH's recommendations for the control of pediculosis in schools include:

- Students found to have evidence of infestation (lice or nits) should be excluded from school attendance until a pediculicide has been applied and all nits have been removed.
- The student should be examined upon returning to school to ensure all nits have been removed.
- The student should be reexamined in ten days to determine if he/she remains free of infestation.

As long as all nits have been removed, a child may return to school the morning after treatment. If only a few nits are found, the nurse may wish to remove them and allow the child to remain in school.

During an outbreak in a school, classroom activities involving frequent body contact between students, such as dancing, wrestling, certain games and group activities should be suspended. To reduce transmission via fomites, hats should be kept in pockets or sleeves; resting mats and pillows should be permanently assigned and kept separated when not in use; and hooks, spaced at least 12 inches apart, should be assigned in cloakrooms.

Each school year an inordinate amount of time and effort is expended by school officials, public health officials and parents dealing with the ongoing problem of pediculosis. Parents must also assume the financial burden of treating and sometimes retreating their children. When schools have an administrative "no nit" policy in place, it makes the nurse's task more realistic and less subjective. It gives the nurse a strategy for returning lice control responsibility to the parents. The end result is an environment of mutual assurance—the nurse is confident that parents are doing everything possible and the parents are assured that their child reenters a school that supports the most comprehensive program possible.

In light of these factors, DOH strongly recommends that schools adopt a "no nit" policy to reduce and control the occurrence of pediculosis. The department recommends this "no nit" policy even though product information and other authoritative sources may not.

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Treatment of Tuberculosis and Tuberculosis Infection in Adults and Children

Bureau of Tuberculosis Control

In May 1993, the Centers for Disease Control and Prevention (CDC) and the American Thoracic Society (ATS) published new guidelines for the treatment of tuberculosis (TB) disease and infection[1]. The new treatment statement, published in the *American Journal of Respiratory and Critical Care Medicine*, replaces the statement issued in 1986.[2] The major changes and additions are summarized below; a comparison with the 1986 statement follows in italics.

Note: this is a summary of **changes** in the new guidelines, not a summary of the guidelines themselves.

Treatment of Tuberculosis

1. Four-Drug Regimen for Initial Therapy

A four-drug regimen is recommended for the initial therapy of TB, except when there is little possibility of drug resistance (i.e., there is less than four percent primary resistance to isoniazid in the community and the patient has had no previous treatment with anti-tuberculosis medications, is not from a country with a high prevalence of drug resistance and has no known exposure to drug-resistant TB).

Previously, a three-drug regimen was recommended for initial therapy, and the addition of Ethambutol was recommended only if isoniazid resistance was suspected. Also, the previous statement recommended daily therapy for the first two months followed by daily or twice-weekly therapy. The new statement gives two more options: 1) twice-weekly therapy after an initial two-week period of daily therapy ***or*** *2) fully intermittent therapy (three-times-weekly therapy from the beginning) when four drugs are given for the entire six months of treatment.*

2. Six-Month Regimen for HIV-Infected Persons

The recommendations for the duration of treatment for TB in HIV-infected persons are the same as for persons not infected with HIV. However, in HIV-infected patients it is critically important to assess the clinical and bacteriologic response to therapy. If there is evidence of slow or suboptimal response, therapy should be prolonged as judged on a case-by-case basis.

Treatment for HIV-infected persons was not discussed in the 1986 statement, but in other statements since 1986, CDC has recommended longer therapy for HIV-infected persons. Recent data indicate that most HIV-infected persons respond well to short-course therapy. However, longer therapy is recommended for TB infection in HIV-infected persons (see below).

3. More Widespread Use of Directly Observed Therapy

Consideration should be given to treating all patients with directly observed therapy.

Directly observed therapy was specifically recommended only for persons whose treatment had failed or who had relapsed, although ensuring adherence was emphasized for all patients.

4. Minimum of 12 Months of Therapy for Children with Some Forms of Extra-pulmonary TB

In general, extrapulmonary TB should be managed according to the principles and with the drug regimens outlined for pulmonary TB. In children, however, a minimum of 12 months of therapy is recommended for miliary TB, bone and joint TB or tuberculous meningitis.

Previously, it was stated that longer therapy may be necessary for lymphadenitis and bone and joint TB. A 12-month regimen for children was not specified.

5. A Four-Month Regimen for Smear- and Culture-Negative TB

A four-month regimen of isoniazid and rifampin, preferably with pyrazinamide for the first two months, is acceptable therapy for adults who have active TB and who are sputum smear and culture negative, if there is little possibility of drug resistance.

No specific regimen for smear- and culture-negative TB was recommended in the 1986 statement.

6. Fixed Drug Combinations to Enhance Adherence

The use of fixed drug combinations is strongly encouraged in adults to enhance patient adherence and reduce the risk of inappropriate monotherapy.

This was not mentioned in the previous statement.

7. Regimens for Isoniazid-Resistant TB

For TB resistant only to isoniazid, a six-month regimen of isoniazid, rifampin, pyrazinamide and either ethambutol or streptomycin is effective. When isolated isoniazid resistance is documented, isoniazid should be discontinued and pyrazinamide should be continued for the entire six months of therapy. When isoniazid resistance is documented in the nine-month regimen without pyrazinamide, isoniazid should be discontinued. If ethambutol was included in the initial regimen, treatment with rifampin and ethambutol should be con-

A copy of the new ATS/CDC guidelines for the treatment of tuberculosis disease and infection can be obtained from your local county health department or by contacting the Bureau of Tuberculosis Control at (314) 751-6122.

tinued for a minimum of 12 months. If ethambutol was not included initially, susceptibility tests should be repeated, isoniazid should be discontinued and two new drugs (e.g., ethambutol and pyrazinamide) should be added.

Only the regimen of rifampin and ethambutol for 12 months was discussed in the previous statement.

8. Multidrug-Resistant TB

Multidrug-resistant TB (i.e., TB resistant to at least isoniazid and rifampin) presents difficult treatment problems. Treatment must be individualized and based on susceptibility studies. In such cases, consultation with a medical expert is recommended. Because of the poor outcome in such cases, it is preferable to give at least three new drugs to which the organism is susceptible. The regimen should be continued at least until bacteriologic sputum conversion is documented, followed by at least 12 months of two-drug therapy. Often a total of 24 months of therapy is given empirically. The role of new agents, such as the quinolones and amikacin, in the treatment of multidrug-resistant disease is not known, although these drugs are commonly being used in such cases. Finally, surgery appears to offer considerable benefit and significantly improves cure rates for patients in whom the bulk of disease can be resected.

Multidrug-resistant TB was not addressed in the 1986 statement. Of note, many clinicians recommend at least 18–24 months of multidrug therapy after culture conversion.

Treatment of Tuberculosis Infection

1. Treatment of TB Infection in Children

For infants and children younger than four years old who have no other risk factors, a reaction of ≥10 mm is considered positive. The American Academy of Pediatrics recommends that children receive nine months of therapy for TB infection.

The different cutpoints were not specifically discussed in the previous statement. Also, this is the first time that children younger than 4 years old who have no other risk factors have been mentioned specifically; they were not included in the 10-mm cutpoint group in the 1990 Diagnostic Standards and Classification of TB.

2. Treatment of TB Infection in HIV-Infected Persons

For adults and children with HIV infection, a reaction of ≥5 mm is considered positive. Persons with a positive skin test result and HIV infection should be considered for preventive therapy regardless of their age. Anergic HIV-infected adults at increased risk for TB should also be considered for preventive therapy. Twelve months of preventive therapy is recommended for adults and children with HIV infection and other conditions associated with immunosuppression.

In the previous statement, preventive therapy was recommended for HIV-infected persons; however, the duration of preventive therapy, as well as a specific cutpoint for the tuberculin skin test results, was not discussed.

3. Regimens for Persons With an Abnormal Chest Radiograph or Silicosis

In patients who have a positive tuberculin skin test result and either silicosis or a chest radiograph demonstrating old fibrotic lesions, and who have no evidence of active TB, acceptable regimens include 1) four months of isoniazid plus rifampin **or** 2) 12 months of isoniazid if infection with drug-resistant organisms is judged to be unlikely.

The four-month regimen is a new recommendation based on recent studies.

4. Directly Observed Preventive Therapy

As with treatment for TB, the success of preventive therapy depends on whether patients adhere to the prescribed regimen. Although not evaluated in clinical studies, directly observed preventive therapy may be used for at-risk adults and children who cannot or will not reliably self-administer therapy.

Directly observed preventive therapy was not mentioned in the 1986 statement.

5. Treatment of TB Infection in Pregnant Women

Although no harmful effects of isoniazid to the fetus have been observed, preventive therapy is generally delayed until after delivery. There does not appear to be any substantial increase in TB risk for women as a result of pregnancy. However, for pregnant women who are likely to have been recently infected or who have high-risk medical conditions, especially HIV infection, isoniazid preventive therapy should begin when the infection is documented.

In the previous statement, preventive therapy was recommended for newly infected pregnant women after the first trimester; preventive therapy for HIV-infected pregnant women was not discussed.

6. Monitoring During Preventive Therapy

All persons receiving preventive therapy should be questioned at monthly intervals about symptoms of adverse reactions. In addition, in person 35 years and older, hepatic enzymes should be measured before the start of isoniazid preventive therapy and monitored monthly throughout treatment. The factors associated with an increased risk for hepatitis include age greater than 35, the daily use of alcohol, chronic liver disease and injection drug use. Also, some evidence suggests that black and Hispanic women are at greater risk for fatal hepatitis.

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Hantavirus

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The latest of the emerging pathogens is a new variant of hantavirus. This type of organism reiterates the fact that the ecology of disease is complex, often intertwining its way through a number of animal species, utilizing vectors and following pathways of infection that are intricate and not fully understood by the scientific, medical or public health communities.

In 1993, an epidemic of acute respiratory illness with high mortality occurred in the southwestern United States. It baffled medical science for a short time. Through the combined efforts of epidemiologists, microbiologists and molecular biologists, the Centers for Disease Control and Prevention (CDC) isolated the causative agent in a little over 30 days. The last time an agent from this virus group caused an outbreak, it took scientists over 20 years to find the causative agent.

From the occurrence of the first case in May 1993 through October 5,1994, there have been 94 laboratory-confirmed cases of hantavirus infections reported from 20 states, with a mortality rate of 53.2 percent. See Figure 1.

The hantavirus family is composed of four commonly-known viruses: Hantaan, Seoul, Puumala and Prospect Hill. These viruses, which cause renal diseases with hemorrhagic fever, primarily occur in Asia and Europe. The only member of the hantavirus family that had previously been found in the United States was the Prospect Hill virus, but it had not produced disease in man. See Table 1.

The newly discovered hantavirus has been named Muerto Canyon. Unlike the other members of the hantavirus family, this virus causes an acute respiratory condition, now called Hantavirus Pulmonary Syndrome (HPS). Recognition of HPS during its early stages is difficult because of the nonspecificity of symptoms. Later symptoms include tachypnea, hemoconcentration, thrombocytopenia and leukocytosis with a high proportion of band cells and other features. Prompt control of rapidly progressive hypoxia, avoidance of excessive fluid administration and the early use of inotropic and pressor drugs are important in treating HPS.

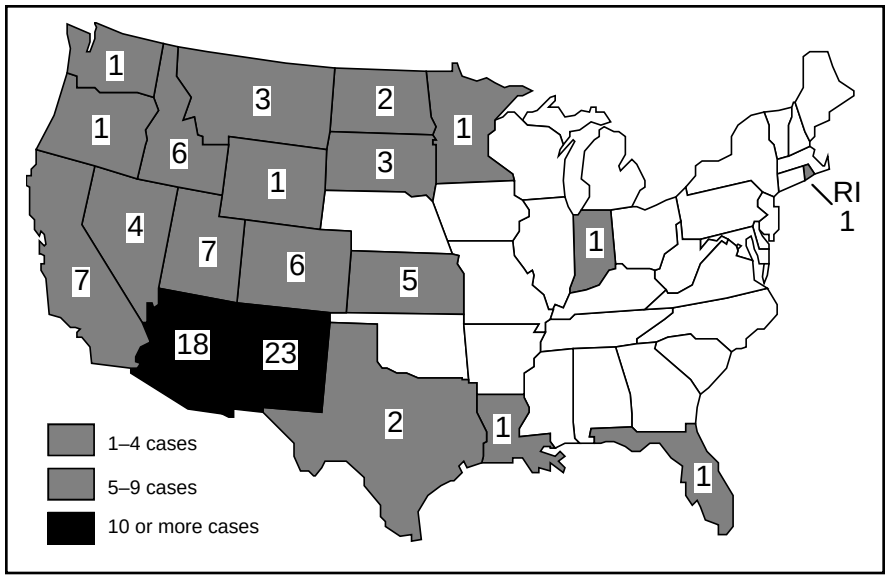

Figure 1. Hantavirus pulmonary syndrome cases by state of residence, United States, October 5, 1994.

Table 1. Hantaviruses Identified From Rodents, United States, 1993

Virus	Host	Disease
Seoul	*Rattus* spp.	Hemorrhagic Fever Renal Syndrome (mild)
Prospect Hill	*Microtus pennsylvanicus*	None
Muerto Canyon	*Peromyscus maniculatus*	Hantavirus Pulmonary Syndrome

To date, hantaviruses have not been reported in Missouri. The CDC's theory, based on the testing of the white-footed prairie mouse, is that wherever you find this species you will find hantavirus. The distribution of this species, *Peromyscus maniculatus*, ranges throughout most of the United States with the exception of the extreme southeastern part of the country. This suggests the vector is present in Missouri. Does this mean the disease will follow? Possibly, but not necessarily. There are steps in the transmission cycle that are still not known or understood. If the presence of the mouse, the virus and casual contact were the only requirements for disease causation, there would be thousands of cases of hantavirus in the United States rather than 94 in the past year.

The best public health method for prevention of hantavirus is to follow the age-old practice of rodent control and rodent-proofing the human habitat. This recommendation will be the same wherever and whenever HPS is diagnosed and until more is known about the transmission of hantavirus.

Hantavirus Points to Remember

What is Hantavirus Pulmonary Syndrome (HPS)?

It is a hantavirus which causes respiratory disease and is carried by wild rodents such as the white-footed prairie mouse, *Peromyscus maniculatus*. Mice do not appear ill while carrying the hantavirus.

How does a person get HPS?

It is thought that individuals become infected with HPS after breathing airborne particles of urine, droppings or saliva from infected rodents. Most cases in the United States have been associated with occupying rodent-infested vacant cabins or other dwellings, cleaning barns or other out buildings, disturbing rodent-infested areas while hiking or camping, planting or harvesting fields, and living in or visiting areas where there has been an increased rodent population. The virus may be spread by handling infected rodents, their nests or droppings, and then touching the nose, mouth or eyes. No evidence of person-to-person spread exists.

Who can be affected by HPS?

Anyone can get HPS. The illness has occurred in whites, Hispanics and Native Americans ranging in age from young to middle age adults. No young children or elderly have been reported with the illness to date.

Can animals transfer HPS?

Cats and dogs are not known to spread hantavirus from rodents to people. Predators, such as snakes, hawks, owls and coyotes, help control rodents and are not believed to spread the disease.

What are the symptoms of HPS?

The first symptoms, appearing one to six (usually two to three) weeks after contact with the virus, are flu-like and may include: fever, muscle and body aches, chills, dry cough, headaches, nausea, vomiting and diarrhea. The lungs will then begin to fill with fluid, making breathing difficult. If you have been exposed to rodents and experience these symptoms, seek medical attention immediately.

How is a diagnosis of HPS made?

Blood sera and other tissue samples should be submitted to the Centers for Disease Control and Prevention for testing through the Missouri State Public Health Laboratory.

How should I get rid of dead rodents, droppings or nests?

Removing rodents from your home decreases the risk for HPS. We recommend following these standard rodent-removal and cleanup guidelines:

- Set spring traps that will kill mice.
- Always use rubber gloves when handling dead rodents, droppings or nests.
- Spray the nest or dead rodent until soaked with a household disinfectant solution of three tablespoons of bleach in one gallon of water. Other disinfectants can be used as directed. Let the area soak thoroughly for 10–15 minutes.
- Remove the nest or rodent using a long-handled shovel or rubber gloves.
- Double-bag the rodent or nest securely with plastic bags and dispose of in the trash. Persons in rural areas may bury the waste three to five feet deep.
- Clean up the rodent area and traps by spraying with the disinfectant solution. Let the area soak for 10–15 minutes. While still wearing gloves, wipe up the area with paper towels or rags. Double-bag all paper towels, rags and gloves used in the cleanup. Dispose of them in a tightly covered trash container.
- Wash your hands with soap and water after completing the cleanup.

How should I clean my home after rodents are removed?

Floors, countertops, cabinets and other surfaces should be disinfected with a solution of three tablespoons of household bleach in one gallon of water, or with a commercial disinfectant. Do not sweep floors with a broom, or vacuum until the area has been disinfected. Rugs can be steam cleaned. Dirt floors should be sprayed with a disinfectant solution.

How can I prevent rodents from entering my home?

- Seal, cover or screen all holes in walls or floors larger than one-quarter inch.
- Keep food (including pet food) and water covered and stored in rodent-proof metal or thick plastic containers with tight-fitting lids.
- Clean up spilled food.
- Store garbage in rodent-proof metal or thick plastic containers with tight-fitting lids. Keep containers at least 12 inches off the ground.
- Place three inches of gravel under the base of mobile homes to discourage rodent burrowing.
- Place woodpiles 100 feet or more away from the house and elevate wood at least one foot.
- Remove any food sources near buildings that might attract rodents.

1993–94 Arboviral Disease Surveillance

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THE MEANING OF THE FLOODS OF 1993, THE ARBOVIRAL PERSPECTIVE

1993 Mosquito Surveillance

The Flood of 1993 was predicted by many to produce large increases in mosquito populations and an increase in the number of mosquito-borne diseases.

Through cooperative agreements with the Centers for Disease Control and Prevention (CDC) and federal emergency funds, five entomology-surveillance teams worked seven days a week from August 4 through October 1, 1993. Dividing the Mississippi and Missouri flood plain into five regions, the five two-man teams set 180 night traps per week. Through the mosquito season, which ended in October, 1,759 pools of mosquitoes, including 123,863 individual mosquitoes, were checked for St. Louis encephalitis. These specimens were analyzed by both CDC in Fort Collins, Colorado and by Dr. Christine Frazier at Southeast Missouri State University in Cape Girardeau, Missouri, using ELISA antigen capture test. The results indicated that St. Louis encephalitis viral activity was not occurring in the mosquito populations surveyed in Missouri.

Similar surveys conducted in the eight other states affected by the flood also confirmed little or no St. Louis encephalitis activity occurring in those states. The Illinois Department of Health's wild bird surveillance program reported two juvenile birds out of 2,795 birds positive for St. Louis encephalitis. Two human cases of St. Louis encephalitis were serologically diagnosed in Illinois in 1993, one case in Whiteside County in the northeastern part of the state and one case in Cook County in the Chicago area. This was the only St. Louis encephalitis activity that was discovered in any species in the nine states affected by the floods.

In addition to mosquito surveillance, an active surveillance for St. Louis (SLE), western equine (WEE), California (CE) and LaCrosse (LCE) encephalitis in equine and human populations was conducted. This surveillance included weekly contact by telephone with predesignated sentinel veterinarians, physicians and hospitals located throughout the state. None of the three active-surveillance systems reported any arboviral activity in Missouri.

While flood-related arboviral activity was not detected in Missouri in 1993, it cannot be a forgone conclusion that danger from arboviruses does not exist. Epidemiological and entomological intelligence tells us that for the past decade arboviral activity has been low in Missouri (zero to two cases per year) and in the Midwest. It also tells us that major outbreaks of arboviral disease can occur two to seven years after a major flood. There is no doubt that arboviruses are present, as sporadic disease cases continue to occur. Time is needed for the virus to amplify in mosquitoes and lower vertebrae, such as wild birds, before the level of virus activity is high enough to detect and cause disease outbreaks in equine and humans, which normally serve as the dead end hosts for the disease.

The mosquitoes that are capable of transmitting arboviruses are called vector mosquitoes. Specifically, the mosquito that transmits St. Louis encephalitis is the *Culex pipiens*, with *Culex tarsalis* the transmitter of western equine encephalitis. Entomological knowledge of the ecology of the habitats of these mosquitoes is essential in conducting mosquito-control programs. Both of these mosquitoes breed in stagnant water, such as those left by receding flood waters. Large pools of stagnant water do not lend themselves to larvicidal control as the amount of water to cover is too great. The best method of vector mosquito control in these situations is adulticiding and eliminating these stagnant breeding grounds.

Mosquito surveillance is of utmost importance in determining if these vector mosquitoes are indeed carrying any arboviruses. Nightly collection and expeditious analysis is crucial as only early detection allows time for mosquito-control programs to be effectively instituted. It is generally accepted that from the time arboviruses are detected in mosquito populations and wild birds, there is only a two-week period before enough virus amplification occurs to cause disease in the equine and/or human populations.

Depending on the number of vector mosquitoes detected, the number of those mosquitoes carrying an arbovirus and the human population at risk, determinations are made as to what level of mosquito control should be instituted. Mosquito-control programs in Missouri could entail limited ground application, ground vehicle application or limited aerial spraying. Should the magnitude of the affected area involve several municipalities or counties, the federal government would become involved in managing and funding the mosquito-control efforts.

California, LaCrosse and Western Equine Encephalitis

Although no human or equine cases of St. Louis or western equine encephalitis were detected in Missouri in 1993, a human case of LaCrosse encephalitis in Stone County was diagnosed from autopsy specimens. LaCrosse encephalitis is not a flood-water disease entity. It is spread by *Aedes triseriatus*, a mosquito that breeds in tree holes that contain water. It can be found throughout southern Missouri. Twelve sporadic, human California and LaCrosse encephalitis cases are the only mosquito-borne encephalitis cases that have occurred in Missouri within the past 15 years. Three cases of western equine encephalitis were detected in equine during the same time period. See Table 1. Figure 1 shows the location of human encephalitis cases in Missouri from 1979–93.

Table 1. Reported Encephalitis Cases by Type, Year and County, Missouri, 1979–93

	Human Cases California		Equine Cases Western Equine (WEE)
1979	1	Buchanan	0
1980	1	Oregon	0
1981	0		0
1982	5	Howell (3), Greene (2)	0
1983	1	Boone	0
1984	2	Texas, Reynolds	0
1985	0		0
1986	1	Taney	0
1987	0		0
1988	0		1 Pettis
1989	0		0
1990	0		0
1991	0		0
1992	0		2 Johnson
1993	1	Stone (LaCrosse)	0
Total	**12**		**3**

1975 - St. Louis Encephalitis Outbreak

Missouri	=	3 Cases	1 Death
Illinois	=	578 Cases	47 Deaths

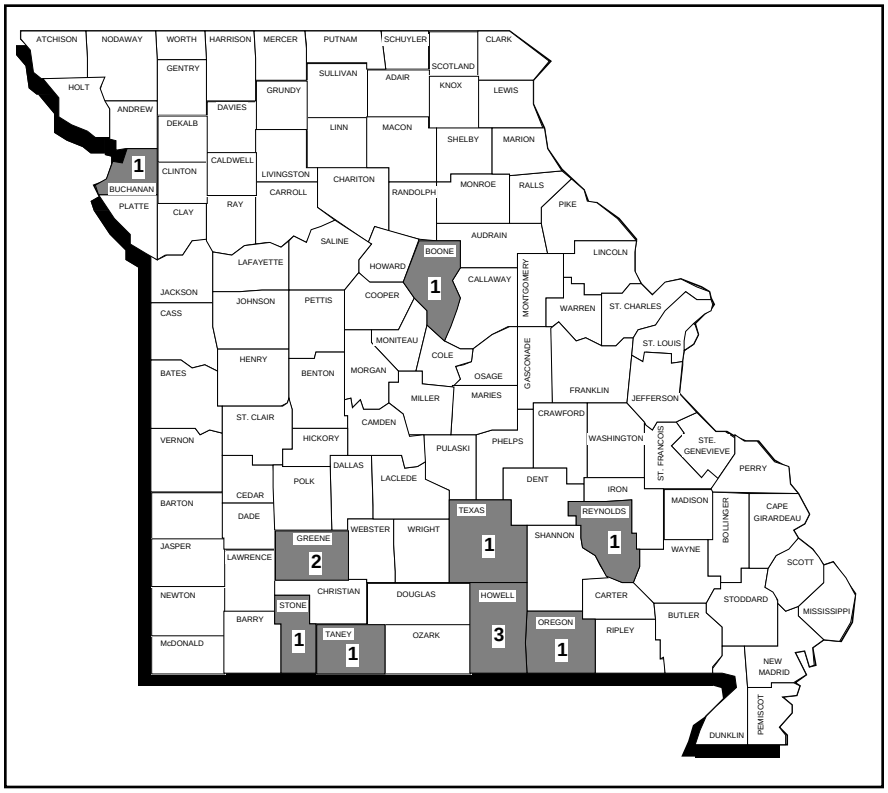

Figure 1. Reported human California encephalitis cases by county, Missouri, 1979–93.

Prevention Strategies

The ecology of the disease and the environment must be understood both to predict disease occurrence and to institute measures to prevent occurrence in equine and human populations.

Not all mosquitoes are capable of transmitting disease. The mosquitoes that appear in largest numbers due to heavy rains, flash flooding or rising waters are of the nuisance variety. They are not physically capable of spreading arboviruses. The ecology of early, fresh-water flooding not only produces ideal conditions for mosquitoes, it also produces ideal conditions for the predatory insects and fish life that feed on these increased numbers of nuisance mosquitoes. Nature has a built-in balance system. Birds and insectivorous bats also feed on these increased mosquito populations. Other vertebrae animals, such as equine, either vacate the areas of high mosquito populations or they tolerate their pesky biting. Humans would benefit by following these examples.

The best protection strategies for humans against arboviral diseases are:

- Stay away from river bottoms and flood plains during hot, muggy summer nights. Avoid previously flooded areas during the early evening hours (the main feeding time for mosquitoes).
- For personal protection, wear light-colored clothing that covers as much of your body as possible.
- Use mosquito repellents containing DEET.
- Stay indoors and rid the interior of mosquitoes, either through physical means (tight-fitting screens) or with insecticides (pyrethrins).

While all of these protective measures may seem trite and involve personal responsibility, they are the most effective, economically feasible and environmentally safe. These methods along with sleeping under mosquito nets have allowed many people to survive mosquito-borne malaria in the tropics.

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The alternative to such personal-protective measures is for some governmental agency to take on the task of mosquito control by the application of insecticides. Unfortunately, insecticides applied over a large area not only kills the mosquitoes for several hours, days or a week, depending on the extent of the area sprayed and frequency of application, but also destroys all other insects. This includes the good insects and the predatory insects, thus disturbing nature's ecological balance and placing man in the future equation of continued mosquito control.

The rhetorical question that arises here is how much money should any level of government spend to preclude an annoyance or nuisance, especially when that nuisance is alleviated only for short periods of time unless insecticides are reapplied? Few state governments have become involved in nuisance mosquito control. Missouri has not and likely will not get involved in the future, based on economic considerations.

1994 Mosquito Surveillance

With federal funding, the Department of Health through the Vector-Borne Disease Coordinator in the Bureau of Veterinary Public Health, conducted surveillance programs for St. Louis, western equine, California and LaCrosse encephalitis during the 1994 mosquito season. The following surveillance systems were instituted:

- Active surveillance for human cases of disease
- Active surveillance for equine cases of disease
- Active surveillance for virus activity in mosquitoes
- Active surveillance for virus activity in wild birds
- Monitoring of sentinel chicken flocks for virus activity

These surveillance programs have been established by contracting with institutions in the state to perform various activities:

- The Department of Health placed five sentinel chicken flocks strategically throughout the state, one each in Clay, Vernon, Pettis, Marion and Jefferson counties. Contracts for the maintenance and bleeding of the birds were made with individuals in those counties.
- The serological analysis for the detection of virus activity in the humans, equine, and sentinel chicken flocks was contracted to the Veterinary Medical Diagnostic Laboratory at the University of Missouri in Columbia.
- The surveillance of wild birds was contracted to the United States Department of Agriculture, Wildlife Damage Control Unit, in Columbia.
- The entomology personnel who conduct the mosquito surveillance and the virology laboratory that conducts analysis of mosquitoes were contracted to the Virology Laboratory at Southeast Missouri State University in Cape Girardeau.

All surveillance programs operated from June to October 1994. To date, no disease activity has been detected. Analysis of data collected is being completed and will be published in a future issue of the *Missouri Epidemiologist*.

Parvovirus B-19

(continued from page 13)

Studies show that 50 percent or more of adults have serologic evidence of past infection. Without knowing the prior serologic status, it is expected that less than one percent of pregnant teachers would experience an adverse fetal outcome during an outbreak of parvovirus B19 infection. This risk is probably lower in a nonepidemic setting. Most instances of fetal death associated with B19 infection occurred during the first or second trimesters. From a public health standpoint, it is unwarranted to exclude pregnant women from the school setting during outbreaks of B19. However, on an individual basis, pregnant women should consult their physicians when faced with this type of risk. Because B19 has not been associated with congenital anomalies, there is no reason to consider interruption of the pregnancy.

Although they may not be very comforting to the pregnant teacher or school employee at risk of exposure to B19, there are several reassurances which can be given: careful hygiene will most likely reduce the risk; over 50 percent of adults are immune; even when pregnancy is complicated by infection, most often there will be no ill effects on the fetus.

REFERENCES

1. Benenson AS, ed. Control of communicable diseases in man. 15th ed. Washington, DC: APHA, 1990.
2. American Academy of Pediatrics. Report of the Committee on Infectious Diseases. Elk Grove Village, Illinois: AAP, 1991.
3. Pickering LK, Reves RR. Occupational risks for child-care providers and teachers. JAMA 1990;263: 2096–97.
4. Galpin JE. Interuterine infection with parvovirus B19. Infectious Disease Alert 3/1/92:63–64.

Treatment of TB

(continued from page 17)

Persons taking certain medications concurrently with isoniazid may be at increased risk for hepatitis or drug interactions. More careful monitoring should be considered for these groups; this may include more frequent laboratory monitoring.

In the previous statement, laboratory monitoring was recommended only for persons 35 years of age and older.

REFERENCES

1. American Thoracic Society. Treatment of tuberculosis and tuberculosis infection in adults and children. Am J Respir Crit Care Med 1994; 149: 1359–74.
2. American Thoracic Society. Treatment of tuberculosis and tuberculosis infection in adults and children. Am Rev Respir Dis 1986; 134:355–63.

Illusion of an Epidemic

(continued from page 10)
four erythrogenic exotoxins A, B, C or D—proteins that are pyrogenic and enhance susceptibility to lethal shock by endotoxin. There are over 80 M protein types, but all share certain common features. The M protein contains a fibrinogen-binding region; binding of the host protein to the surface of GAS inhibits complement binding, thus impairing phagocytosis (Infec Immune 57:29, 1989). The M type-1 serotype is associated especially with invasive disease, and there is evidence to suggest that the proportion of M serotypes has been increasing in the U.S. (Lancet 336:1167, 1990). The GAS organism is a virtual factory for the production of extracellular proteins as well. Over 20 such products have been identified, many of which allow the bacteria to spread rapidly and destroy soft tissues e.g., hemolysins, streptolysin O, streptolysin S, hyaluronidase, DNAse, and streptokinase.

The best preventive advice available, regarding invasive GAS with necrotizing fasciitis is proper wound treatment and management. Persons with wounds should take appropriate measures to keep the wounds clean and should seek prompt medical attention if signs of infection occur.

Head Lice

(continued from page 15)
Films and literature regarding head lice are available through the Department of Health. A listing of materials and order blanks are in the DOH Audiovisual and Literature Catalog, which has been sent to each school. For more information regarding prevention and control of pediculosis, call the **Bureau of Communicable Disease Control at (800) 392-0272**.

You may also call the **National Pediculosis Association at (800) 446-4672** for information and to report adverse reactions to a pediculicide.

Missouri Perinatal Association Annual Conference

March 23–24, 1995

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Ephedrine Becomes Controlled Substance in Missouri

Reprinted from a Department of Health news release issued on August 29, 1 994.

Ephedrine, an over-the-counter stimulant that has been a fad among teens, is now a controlled substance in Missouri. Many products that contain ephedrine will only be available with a prescription because of a new law that took effect August 28, 1994.

Ephedrine abuse has become a recent fad among teens seeking a stimulant effect or weight loss. Products containing ephedrine that are advertised as stimulants or for weight loss are widely available in convenience stores.

Ephedrine is particularly dangerous because young people often take the drug in greater quantities than the labels recommend. When the drug is overdosed, some very serious side effects can happen, including agitation, nervousness, insomnia, fear, irritability, breathing difficulties, increased heart rate and increased blood pressure. An overdose could have very serious, if not fatal, effects in persons with existing heart or blood-pressure conditions.

Ephedrine obtained in an over-the-counter tablet form is also the key ingredient used in clandestine labs to make methamphetamine and methcathinone, or CAT. CAT is a highly addictive, potent stimulant that is easily made in a laboratory garage, basement or apartment. The chunky, off-white powder somewhat resembles crack cocaine in appearance and potency.

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Alternate forms of this publication for persons with disabilities may be obtained by contacting the Missouri Department of Health, Division of Environmental Health and Epidemiology, P.O. Box 570, Jefferson City, MO 65102, (314) 751-6128. TDD users can access the preceding phone number by calling (800) 735-2966.

This newsletter can be recycled.

Marty Huber Appointed Nurse Consultant for the Bureau of Tuberculosis Control

Dan Ruggiero
Bureau of Tuberculosis Control

Marty Huber, R.N. was appointed by the Department of Health, Bureau of Tuberculosis Control, as Nurse Consultant effective April 1, 1994. Marty, who has been with the Department of Health for over two years, brings to the program a wide variety of nursing and tuberculosis experience. Ms. Huber received her B.S.N. degree from Arizona State University. She is currently enrolled in the Masters of Public Health Program at St. Louis University.

From 1978–82, Marty was the program manager of the Kentucky Tuberculosis Control Program and, more recently, the project coordinator for the Special Invasive Bacterial Infections Study conducted by the Centers for Disease Control and Prevention through the Missouri Department of Health, Bureau of Communicable Diseases. Marty has traveled extensively throughout the country assuming a variety of public health positions ranging from a sexually transmitted disease clinic nurse to a nurse consultant for a lead poisoning control project and a statewide high blood pressure control program. She is the author of several publications and has done extensive teaching on a variety of topics. Prior to coming to the Department of Health, she was an instructor in the Department of Nursing Sciences at Lincoln University in Jefferson City.

As Nurse Consultant for the Bureau of Tuberculosis Control, Marty will be working closely with the bureau chief in coordinating program activities. In her new role, she will provide consultation and technical assistance to public and private health-care providers regarding tuberculosis control policies, guidelines and procedures. Marty will also play a key role in developing and coordinating tuberculosis educational materials, as well as setting up programs and conferences on tuberculosis control throughout the state.

Marty can be reached in the **Bureau of Tuberculosis Control at (314) 751-6122.**

Missouri EPIDEMIOLOGIST

Volume XVI, Number 5 *November–December 1994*

Early Syphilis in the Bootheel Region of Southeast Missouri

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During 1992, seven rural counties in Southeast Missouri, commonly referred to as the "Bootheel," experienced a significant increase in reported early syphilis cases (primary, secondary and early latent under one year duration). Following a quarterly average of 6.75 cases (with a total of 27 cases) during 1991, increases in reported cases began to occur during the first quarter of 1992, and continued throughout the remainder of the year. During the fourth quarter of 1992, a total of 93 cases were reported, which represented an increase of 3,000 percent over the same quarter in 1991. The case rate for 1992 was approximately 96 per 100,000 population.

Extensive screening, treatment and partner notification activities resulted in significant decreases in reported cases from the first quarter of 1993, when 44 cases were reported, to the third quarter of 1994, when 16 cases were reported. The case rate for 1993 was 60.7 per 100,000 population, down from the 1992 rate by 39 percent. The projected rate for 1994, based on data from the first three quarters of the year, is 34 cases per 100,000 population. During the three year period, 1992–94, a total of 345 early syphilis cases were identified. See Figure 1.

The Bootheel region's low socioeconomic status and high unemployment levels, particularly in the minority community, have raised concerns in the past regarding the potential for increased sexually transmitted disease occurrence in the area. The emergence of syphilis outbreaks in association with crack cocaine in many areas of the United States, and the fact that the Bootheel is located on a major crack distribution route between New Orleans and Chicago, have put this part of Missouri at extremely high risk for early syphilis. Approximately 125 (36%) of the 345 early syphilis cases identified in the Bootheel area from 1992 to September 1994 were among drug users or sex partners of drug users. Males often traded drugs for sex, and females used sex to obtain drugs or money for drugs. See Figure 2.

Nearly all of the syphilis cases in the Bootheel are from heterosexual contacts; females accounted for 60 percent of the cases during this outbreak. Most heterosexual syphilis outbreaks have an equal sex distribution. In this instance, however, the large percentage of female

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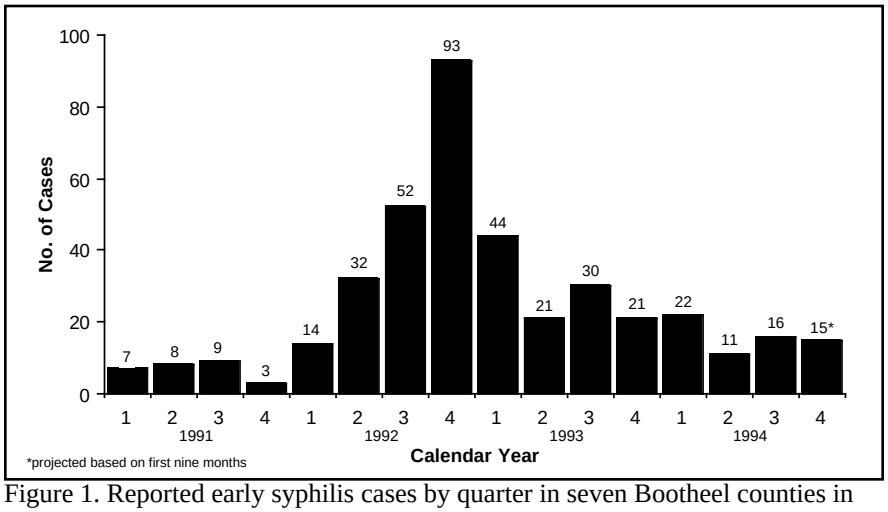

Figure 1. Reported early syphilis cases by quarter in seven Bootheel counties in Southeast Missouri, calendar years 1991–94.

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cases is the result of a significant number of male visitors to the area whose syphilis was diagnosed, treated and reported elsewhere. See Figure 3.

Access to medical care for many of the persons involved in this outbreak was limited, resulting in increased disease transmission because infected individuals did not receive medical evaluation and appropriate treatment early in the course of their infection.

The significant increases in early syphilis in the Bootheel were noted by public health officials in mid-1992 and disease intervention personnel were diverted from other areas of the state to supplement the one Disease Intervention Specialist (DIS) assigned to the 25-county Southeast District. In early 1993, the Centers for Disease Control and Prevention (CDC) assigned four additional DIS to the area for two months. Also, local hospital emergency rooms agreed to provide therapy for diagnosed patients and their sex partners. These actions were major contributing factors in bringing the outbreak under control.

The necessary additional resources to carry out these control measures were provided by redirecting funds from other areas. Also, the Missouri General Assembly provided additional funding to allow assignment of a second full-time DIS to the Southeast District on a permanent basis to provide partner elicitation interviews, sex partner notification and other disease intervention follow up. This action will help keep syphilis in the Bootheel under control by allowing very early intervention activities to occur.

While syphilis has not been eradicated from the Bootheel area, early syphilis remains at a more manageable level during the latter half of 1994. It is imperative that physicians maintain a high level of suspicion relative to syphilis when performing diagnostic examinations. Widespread serologic screening for syphilis should continue to be

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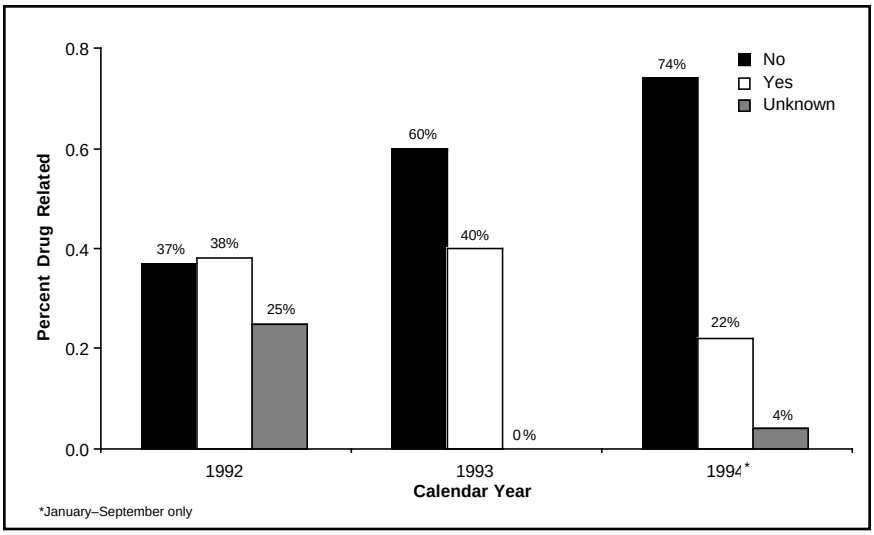

Figure 2. Drug-related early syphilis by calendar year in seven Bootheel counties in Southeast Missouri, calendar years 1992–94.

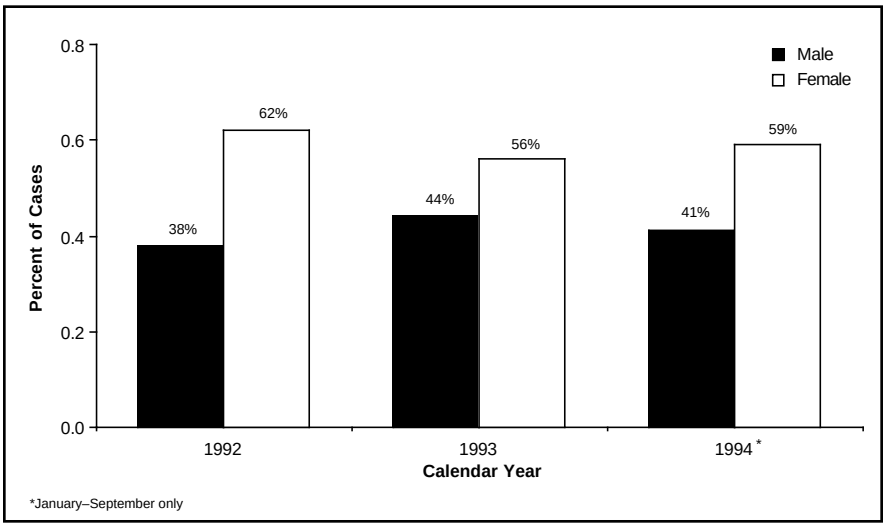

Figure 3. Reported early syphilis cases by sex, Missouri, calendar years 1992–94.

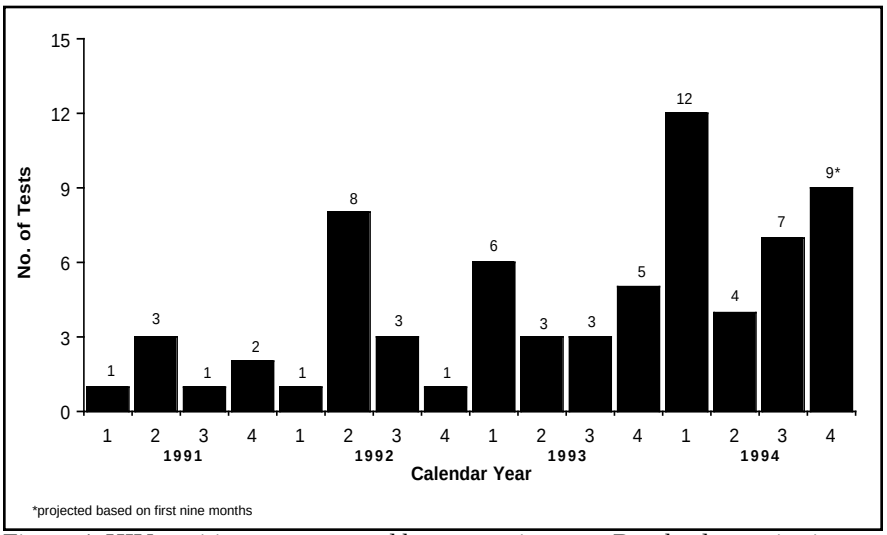

Figure 4. HIV-positive tests reported by quarter in seven Bootheel counties in Southeast Missouri, calendar years 1991–94.

Lead Exposure Study for Big River Mine Tailings Site

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Introduction

The site named The Big River Mine Tailings/St. Joe Minerals Corporation at Desloge, St. Francois County, Missouri is situated within a large area of southeast Missouri known as the "Old Lead Belt," approximately 70 miles south of St. Louis. This area was a major producer of lead in the world. The site's surface is approximately 600 acres, and mine tailings in piles up to 100 feet high are distributed over the site as a result of 30 years of accumulated mining residues.[1] The last mine officially closed[2] in October 1972 and mining operations ceased. Old mines, however, exist underneath the cities of Desloge, Bonne Terre, Elvins, Flat River and Leadwood. Approximately 250 million tons of waste material, most of it contained in the piles, lies in the area.[3]

Based on the information presented on the Preliminary Public Health Assessment for the Big River Mine Tailings site,[4] the Health Activities Recommendation Panel (HARP) at the Agency for Toxic Substances and Disease Registry (ATSDR) has determined that exposure to contaminants exists at the Big River Mine Tailing Site and surrounding areas, and that levels of contaminants found in the area are high enough to cause health problems.

Considering the widespread contamination of soil and dust with lead and given the potential for social costs of lead poisoning in children, and in light of the HARP recommendations, the Missouri Department of Health (DOH) proposed to ATSDR to conduct a two-year study of children exposure to lead. The study will examine blood lead levels among children potentially at high risk of exposure to lead-contaminated soil, surface and ground water and dust at three target areas located near the Big River Mine Tailings Site.

The study will test four hypotheses, that:

1. Children living in a geographically defined area surrounding the Big River Mine Tailings Site have higher blood-lead levels than children living in a non-mining area.
2. Lead contamination of the soil in the study areas is mainly a result of past mining and mill tailing operations. Therefore, the metal content of soil in the yards of homes in the study areas should resemble the metal composition of the chat piles.
3. Lead in soil and dust from mining operations is the primary factor contributing to blood-lead levels.
4. Children with behaviors that are associated with greater ingestion of soil and dust will have higher blood-lead levels than children without these behaviors.

Objectives of the study at the Big River Mine Tailings Site to test these hypotheses include:

1. Measure exposure to lead by analyzing blood from study and control participants.
2. Compare blood-lead levels between study and control groups.
3. Measure environmental sources of lead.
4. Compare environmental lead between study and control areas.
5. Evaluate the relationship between blood-lead levels and environmental sources of lead.
6. Determine the relationship between blood-lead levels and behavioral risk factors.
7. Characterize lead to determine the proportion of soil and dust lead that comes from mining operations, and the proportion that comes from other environmental sources.
8. Model environmental factors to determine the proportional contribution of each factor to blood-lead levels.

Background

In 1980, elevated levels of lead detected in fish downstream of the site were reported by the Missouri Department of Conservation (MDOC). Lead levels in edible fillets ranged from 0.4 ppm to 0.7 ppm. The maximum lead level detected was 1.28 ppm. This prompted a news release, issued by the MDOC and the DOH, warning people about fish consumption in the affected area. Fifty miles down the river, from Leadwood to Mammoth access, residents are still under the advisory.[5] The State of Missouri and St. Joe Minerals Corporation, under a 1981 cooperative agreement, started stabilization activities in the pile. These activities have been partially successful.

The United States Fish and Wildlife Service released the results of their study on the effects of the chat and tailings material on the Big River in 1982. The findings reported elevated heavy metal residues, mainly lead, cadmium and zinc, in all biologicals examined. Species studied consisted of algae, rooted plants, crayfish, mussels and fish.[6]

In 1990, an EPA contractor completed a thorough assessment of the site. Laboratory results indicated that lead levels found in samples taken from the piles ranged from 910 ppm to 13,000 ppm with a mean concentration of 2,215 ppm. These values represent extremely high concentrations compared with background concentrations as low as 64 ppm, and are similar to those reported in a study carried out by the University of Missouri-Rolla.[7] Two residential samples and one taken near a day care center showed very high lead concentrations similar to those reported from the tailings.

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Exposure pathways (i.e., ingestion, inhalation) occurring at the site and target study areas are due to contact with ambient air, indoor dust, soil, water and ingestion of fish. For this study, ingestion of soil, particulate and drinking water will be considered as the more relevant pathway for children. Amount of exposure, duration or frequency have not been established.

Chat and tailing materials are used as fill material, mixed with asphalt for road paving and surface coverage and used for many other house and garden uses. Humans and erosion have spread the material throughout the area. Erosion significantly contributes to downgradient deposition of the contaminated material.

Lead Toxicity and Risk

At low levels of exposure, comparable to those found near the site, toxicity has been described. They include decreased attention span, hyperactivity and lower IQ scores.[8] Lead levels as low as 10 µg/dL have been shown to affect child development.[9–15] At low levels of exposure, signs of lead toxicity include decreased attention span, impaired speech and language deficits and behavior disorders such as hyperactivity and lower IQ scores.[8] Needleman and Gatsonis report that children's IQ scores are related inversely to low levels of lead burden.[15] Several adequately conducted studies provide sufficient evidence that children's cognition is adversely affected by lead.[14,16–26]

Adverse effects of lead on intelligence are persistent across socioeconomic strata, as well as different ethnic and racial groups.[10,27] The ATSDR has estimated that 17 percent of American children have blood-lead levels above 15 µg/dL. Among white children, 7 percent of those with good socioeconomic conditions have elevated lead levels in contrast with 25 percent in poor whites. The estimates for black children are 25 percent among those in good socioeconomic conditions compared with 55 percent among poor blacks.[28] As a consequence, more than half of the black children in preschool ages are potentially at disadvantage before entering school. Among black children in preschool age, the relationship of subclinical lead levels with cognitive and sensorimotor impairment is well documented.[29] These are the basis for considering lead poisoning as the "most serious environmental disease for American children."[30]

Several populations are particularly sensitive to adverse health effects from lead exposure. Pregnant women, fetuses and children are particularly affected by lead. Children with glucose six-phosphate dehydrogenase deficiency (most prevalent among blacks) have greater blood-lead levels than non-deficient children with similar exposure. Children with sickle-cell anemia may be particularly sensitive to neurological effects of lead exposure. Children with pica (ingestion of nonfood substance) are also at greater risk because they ingest more contaminated materials. Also, children with dietary deficiencies in calcium, iron and zinc may be susceptible to the adverse effects of lead. Poverty is a major contributor to the cycle of lead poisoning, therefore, poor and minority children are at higher risk due to their living conditions, unhealthy behaviors and poor nutritional status.

Relevant exposure pathways, that is ingestion and inhalation, for children living in poor areas are due to exposure to contaminated paint, materials, ambient air, indoor dust, soil and probably contaminated water pipes. Lead-based paint continues to be a problem in older homes and, therefore, is a major contributor to lead poisoning. Estimates from the Department of Housing and Urban Development (HUD) indicate that 3.8 million homes occupied by children have high levels of lead in dust and lead-based paint in poor condition.[31]

Methods

A complete census of all households in the study and control areas will be conducted to locate children currently living in these areas. Children between 6 – 72 months of age living in the defined areas for at least 60 days prior to the beginning of the study will qualify for participation in the exposed group. We plan to enroll 250 children in the study group. A control group of 150 children will be taken from an area outside the Old Lead Belt. We used census data to select three areas based upon similarities with the study group.

Investigators will visit each household included in the sample. The investigators will complete a questionnaire that includes information on the child and the household and obtain a venous blood sample. Environmental samples from the home (paint, dust and water) and yard (soil) will be collected for analysis. Several of the sites chosen randomly will be evaluated for the identification of sources of lead to determine the contribution of various environmental sources to soil and dust lead levels.

We plan to begin the census phase of the study in March 1995 followed shortly by sample selection and investigation. The data will be analyzed and a report written for both peer and community comment. A final report will then be provided to ATSDR for public release. The investigators will encourage community participation during each stage of the process.

REFERENCES

1. Environmental Protection Agency. Final Report: Listing site inspection, Big River mine tailings, Volume 1, Desloge, St. Francois County, Missouri. Washington DC:EPA document. TDD #F-07-9004-011, 1991.
2. U.S. Geological Survey. Assessment of water quality in non-coal mining areas of Missouri, Rolla, Missouri: USGS Document No. Water Re-

sources Investigation Report 87-4286, 1988.
3. Ecology and Environment, Inc, Field Investigation Team (E&E). Final Report: Listing site inspection, Big River mine tailings, Vol. I. Desloge, St. Francois County Missouri TDD #F-07 9004 011, PAN #F M00616xa Site #Y60, Project #003 Submitted to Region VII, EPA, October 30, 1991.
4. ATSDR. Preliminary public health assessment for Big River mine tailings site, Desloge, St. Francois County, Missouri. Agency for Toxic Substances and Disease Registry, January 12, 1994.
5. Missouri Department of Health. Missouri Department of Health 1993 Fish Advisory. Jefferson City, Missouri, 1993.
6. Schmitt CJ, Finger SE. The dynamics of metals from past and present mining activities in the Big and Black River watersheds, southeastern Missouri. U.S. Fish and Wildlife Service, Columbia National Fisheries Research Laboratory, Columbia, Missouri, 1982.
7. Wixson BG, Gale NL, Davies BE. A study on the use of chat and tailings from the old lead belt of Missouri for agricultural limestone. A Research Report submitted to the Department of Natural Resources. University of Missouri, Rolla, Missouri, 1983.
8. Ernhardt CB, Landa B, Schnell NB. Subclinical levels of lead and developmental deficits, a multivariate follow-up assessment. Pediatrics 1981;67(6):911-19.
9. Bellinger D, Leviton A, Waternaux C, Needleman H, Robinowitz M. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. N Engl J Med 1987;316:1037–43.
10. Bellinger D, Sloman J, Leviton A, Ravinowitz M, Needleman H, Waternaux C. Low-level exposure and children's cognitive function in the preschool years. Pediatrics 1991; 87: 219–27.
11. Dietrich KN, Kraft KM, Bornshein RL, Hammond PB, Berger O, Succop PA, Bier M. Low-level fetal lead exposure effect on neurobehavioral development in early infancy. Pediatrics 1987; 80:721–30.
12. Needleman H, Schell A, Bellinger D, Leviton A, Allred En. The long-term effects of exposure to low doses of lead in childhood: and 11-year follow-up report. N Engl J Med 1990;322:83–8.
13. Ernhardt CB, Wolf AW, Kennard MJ, Ekhard P, Filipovich HF, Sokol RJ. Intrauterine exposure to low levels of lead: the status of the neonate. Arch of Environ Health 1986; 41:287–91.
14. Lyngbye T, Hansen ON, Trillingsgaard A, Beese I, Grandjean P. Learning disabilities in children: significance of low-level lead exposure and confounding effects. Acta Pediatr Scand 1990;79:352–60,
15. Needleman HL, Gatsonis CA. Low-level lead exposure and the IQ of children. JAMA 1990;263:673-8.
16. Bergomi M, Borella P, Fantuzzi G, Vivola G, Sturloni N, Cavazzuti G, Tampieri A, Tartoni PL. Relationship between lead exposure indicators and neuropyschological performances in children. Dev Med Child Neurol 1989; 31: 181–90.
17. Ferguson DM, Fergussen JE, Horwood LJ, Kinzett NG. A longitudinal study of dentine lead levels, intelligence, school performance, and behavior part II: dentine lead and cognitive ability. J Child Psych Pyschiat 1988;29:793–809.
18. Fulton M, Raab G, Thompson G, Laxen D, Hunter R, Hepburn W. Influence of blood lead on the ability and the attainment of children in Edinburgh. Lancet 1987;1:1221–6.
19. Hansen ON, Trillingsgaard A, Beese I, Lyngbye T, Grandjean P. A neuropsychological study of children with elevated dentine lead level: assessment of the effect of lead in different socio-economic groups. Neurotoxicol Teratol 1989;11:205–13.
20. Hawk BA, Schroeder SR, Robinson G, Otto D, Mushak P, Kleinbaum D, Dwanson G. Relation of lead and social factors to IQ of low-SES children: a partial replication. Am J Ment Def 1986;91:178–83.
21. Hatzakis A, Kokkevi A, Maravelias C, Katsouyanni K, Salaminios F, Kalandidi A, Koutselinis A, Stefanis C, Trichopoulos D. Psychometric intelligence deficits in lead-exposed children. In Smith M, Grant L, Sors A, editors. Lead exposure and child development: an international assessment. Dordrecht: Kluwer Academic Publishers, 1989:211–23.
22. Lansdown R, Yule W, Urbanowicz MA, Hunter J. The relationship between blood-level concentrations, intelligence, attainment and behaviour in a school population: the second study. Int Arch Occup Environ Health 1986;57:225–35.
23. Schroeder SR, Hawk B, Otto DA, Mushak P, Hicks RE. Separating the effects of lead and social factors on IQ. Environ Res 1985;38:144–54.
24. Silva PA, Hugher P, Williams S, Faed JM. Blood lead, intelligence, reading attainment, and behaviour in eleven year old children in Dunedin, New Zealand. J Child Psych Psychiat 1988;29:43–52.
25. Winneke G, Brockhaus A, Ewers U, Kramer U, Neuf M. Results from the European multicenter study on lead neurotoxicity in children: implications for risk assessment. Neurotoxicol Teratol 1990;12:553–9.
26. Yule W, Lansdown R, Miller I, Urvanowicz M. The relationship between blood lead concentrations, intelligence, and attainment in a school population: a pilot study. Dev Med Child Neurol 1981;23:567–76.
27. Dietrich KN, Berger O, Succop PA, Bornchin RL. Lead exposure and the motor developmental status of urban 6 year-old children: the Cincinnati prospective study. Pediatrics 1993;91(2):301–7
28. Atlanta Department of Health and Human Services. The nature and

(continued on page 13)

Outbreak of Measles Among Christian Science Students— Missouri and Illinois, 1994

Reprinted from the Morbidity and Mortality Weekly Report, Vol. 43, No. 25, July 1, 1994, published by the Centers for Disease Control and Prevention, Atlanta, GA.

During April 4–May 17, 1994, the largest measles outbreak in the United States since 1992 occurred among students in two communities that do not routinely accept vaccination. This report summarizes the investigation of and control measures for this outbreak.

The outbreak began in a 14-year-old Christian Science high school student who developed a rash on April 4, two weeks after skiing in Colorado where a measles outbreak was occurring. The student lived with her family in a community associated with a Christian Science college in Jersey County, Illinois, and commuted approximately 30 miles to a Christian Science boarding school (kindergarten through grade 12) in St. Louis County, Missouri. From April 16–May 19, 141 persons with measles (age range: 1–24 years) were reported to the St. Louis County Health Department, and 49 persons with measles (age range: 4–25 years) were reported to the Jersey County Health Department. See Figure 1.

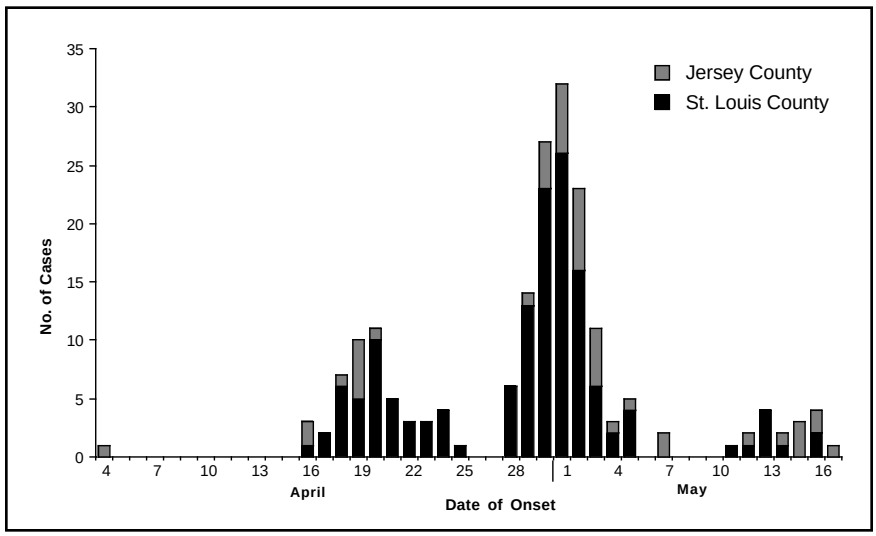


Figure 1. Number of measles cases, by date of rash onset and location, St. Louis County, Missouri and Jersey County, Illinois, April 4–May 17, 1994.

All cases met the measles clinical case definition[1] and were epidemiologically linked to the boarding school and/or college. Fourteen cases were serologically confirmed by detection of immunoglobulin M antibody. All cases occurred among persons not vaccinated before the outbreak. Eighteen prospective students from outside St. Louis County attended a carnival at the boarding school on April 16; eight developed measles after returning home (three to Maine, two to California and one each to Missouri, New York and Washington). Two cases of serologically confirmed measles occurred in persons outside the Christian Science communities. One case occurred in an unvaccinated 35-year-old physician who attended a tennis tournament on April 30 where students from the affected college competed. The other case occurred in a 9-month-old infant who visited a restaurant on April 30 where the college tennis team was eating.

Control measures included offering measles vaccine to students in the affected communities and isolating persons with rashes and those considered susceptible to measles. On April 19, the boarding school and college began isolating persons with rashes in a separate building on each campus and placing 24-hour guards at campus entrances. Only persons with proof of immunity to measles were permitted to enter or leave the campuses. Isolation measures on both campuses remained in effect until 14 days after the appearance of rash in the last persons with measles for each school.

Students who did not live on campus and had no proof of vaccination were voluntarily isolated in their homes, unless they were born before 1957 or could provide documentation of 1) previous physician-diagnosed measles, 2) laboratory evidence of measles immunity, 3) two doses of measles vaccine at least one month apart on or after their first birthday or 4) one dose of measles vaccine on or after April 18, 1994.

Measles vaccination was offered to Christian Science students and persons in the surrounding communities at special clinics offered by the public health departments in both Missouri and Illinois. A total of 149 Christian Science students (K–12) and their siblings were vaccinated in Missouri and 451 in Illinois. Of the 149 students at the boarding school who received measles-mumps-rubella vaccine (MMR) during outbreak control, 61 (41%) developed measles within two weeks after vaccination.

Siblings of persons with measles who were enrolled in public schools in St. Louis County were voluntarily isolated at home. Active surveillance for persons with rashes was initiated in the county public schools on May 9 and consisted of a daily telephone call from the health department to the head nurse in each school district who monitored all student absentees for rash illness. A second dose of measles vaccine was adminis-

tered to 675 students in vaccination clinics conducted in four public schools in St. Louis County and three public schools in the City of St. Louis where rash cases were detected. No outbreak-control vaccination was conducted in Illinois public schools because two doses of measles vaccine had been mandated for all K–12 schoolchildren since 1993, and compliance with this law was considered to be high.

As of June 29, no additional measles cases had been reported among persons outside the Christian Science community in St. Louis County or elsewhere in Missouri or in Illinois. In response to the outbreak, St. Louis County will require two doses of measles vaccine for all schoolchildren by the start of the 1994–95 school year.

Reported by: L Fisher, M Williams, L Feltmann, St. Louis County Dept of Health, Clayton; D Donnell, T Hicks, Missouri Dept of Health. T Macias, L Watson, Jersey County Health Dept, Jerseyville; C Jennings, Illinois Dept of Public Health. National Immunization Program, CDC.

Editorial Note: The magnitude of this outbreak illustrates the potential challenges that groups that do not routinely accept vaccination present for eliminating indigenous measles in the United States by 1996.[2,3] Communities that do not accept vaccination are at risk for recurring outbreaks and may provide foci of infection that can result in further transmission. Measles outbreaks had occurred at both the school and college in this report during 1978, 1980, 1985 and 1989; three students died from measles-related complications in the 1985 outbreak.[2] From January 1 through June 10, 1994, outbreaks among persons who do not accept vaccination in Illinois, Missouri, Nevada and Utah accounted for approximately 50 percent of all reported measles cases (excluding United States territories).

Although Christian Science doctrine does not forbid medical care, many Christian Science parents claim religious exemption from childhood vaccination requirements. Vaccination is accepted by some members, particularly when the consequences of illness are considered less acceptable. During this outbreak, many Christian Science students accepted vaccination to attend school. However, individual decisions to be vaccinated may not be made until an outbreak is established and its potential impact becomes apparent.

During measles outbreaks in educational institutions, revaccination with MMR is recommended.[4] If measles vaccine is administered within 72 hours of exposure, it may prevent or modify illness.[4] The 41 percent postexposure vaccine failure rate in this outbreak underscores the need for a sensitive and timely measles surveillance system to identify cases promptly and to administer vaccine as early as possible. Persons vaccinated more than 72 hours postexposure may develop infection and contribute to further spread of measles.

Factors that may have contributed to limiting this outbreak include the self-imposed isolation of persons with and those susceptible to measles in the Christian Science community, high vaccination levels for one dose of measles vaccine among Missouri students and two doses among Illinois students, cooperation from private physicians in providing a second dose of measles vaccine to school-aged children both before and during the outbreak and media coverage of the outbreak encouraging parents to obtain a second dose of measles vaccine for their children.

The findings in this report illustrate that transmission of measles can be prevented or minimized by 1) maintaining high vaccination levels in the general population, 2) conducting active surveillance in populations that do not routinely accept vaccination and 3) initiating aggressive control efforts during an outbreak. Public health officials should emphasize in communities that do not routinely accept vaccination the importance of vaccination, active surveillance and timely reporting of contagious diseases to the public health department.

References

1. CDC. Case definitions for public health surveillance. MMWR 1990;39 (No. RR-13):23.
2. Novotny T, Jennings CE, Doran M, et al. Measles outbreaks in religious groups exempt from immunization laws. Public Health Rep 1988;103: 49–54.
3. Rodgers DV, Gindler SS, Atkinson WL, Markowitz LE. High attack rates and case fatality during a measles outbreak with religious exemption to vaccination. Pediatr Infect Dis 1993;12:288–92.
4. CDC. Measles prevention: recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR 1989;38(No. SS-9):7.

Readership Survey

The survey form included in the March–April issue was returned by 74, or 0.7%, of approximately 10,700 newsletters mailed. Although the number of returns was small, we appreciate the input and comments from those who took the time to respond.

Slightly more than 90% of the respondents indicated they read more than half or almost all of the articles including 46% who indicated they read almost all of the articles. The newsletter was given an overall rating of excellent by 68% of the respondents. This was a ten point increase from the survey done in 1990. Accuracy was rated excellent by 59% compared to 52% in 1990 and very little change in rating was given for writing style, readability or appearance. The length of articles was rated appropriate by 88%, which is very similar to the 87% rated appropriate in 1990.

The types of articles given excellent ratings were outbreak reports with 69% excellent rating, statistical tables with 45%, environmental with 43% and guidelines with 39%. These were all higher ratings than the same topics were given in 1990. Laboratory reports and outside contributions were rated about the same in both years with 34% and 27% excellent respectively. Reprints and calendar items rated lowest and both somewhat lower in 1994 than 1990.

Several requests for articles will be met in forthcoming issues.

Antimicrobial Resistance Alert

E. Dale Everett, M.D.
Division of Infectious Diseases
University of Missouri Hospital and Clinics

Most of us have practiced during the period of rapid development of many potent and effective antimicrobial agents. Despite these developments, today one can die of microbial infections resistant to all of our current drugs. This phenomenon is **increasing** throughout the world and includes the United States. It is believed by many that a crisis is occurring and will continue to develop in the next few years. Furthermore, discovery of new antimicrobial agents with unique modes of action has slowed precipitously.

Some of the current problems include:

- *Streptococcus pneumoniae* (pneumococcus) resistant to penicillins and cephalosporins (at University of Missouri Hospital and Clinics (UMHC): 7% and 0% respectively).
- Enterococci with high level resistance to aminoglycosides or vancomycin or ampicillin.
- Methicillin-resistant *Staphylococcus aureus* or coagulase-negative staphylococcus (at UMHC: 30% and 55% respectively).
- *Streptococcus pyogenes* (Group A beta-hemolytic streptococci) resistant to erythromycin (at UMHC: 27%).
- *Pseudomonas* species resistant to all antibiotics.
- Multiple outbreaks of a variety of gram-negative rods resistant to ceftazidime, cefotaxime, imipenem, aminoglycosides, etc.
- Development of multiple-antibiotic resistance *in vivo* by *Enterobacter* species during ceftazidime or cefotaxime therapy.

Possible solutions to prolong the usefulness of currently available antibiotics are:

- Make a **habit** of washing hands between each patient examination.
- Reduce inappropriate usage of outpatient antimicrobials for upper respiratory infections.
- Educate patients that the vast majority of respiratory infections are viral and do not respond to antimicrobials.
- Use the narrowest spectrum antimicrobial for the infection diagnosed.
- Minimize usage of vancomycin, especially for antibiotic-induced diarrhea (use metronidazole instead).
- Use prophylactic antimicrobials judiciously. Most surgical cases can be managed with a single well-timed preoperative dose.
- Reserve multi-antibiotic regimens for those situations in which it has been reported that two or more antibiotics are preferred to a single agent (e.g., piperacillin plus gentamicin for *Enterobacter* species).
- Avoid ceftazidime or cefotaxime as therapy for *Enterobacter* species, regardless of lab sensitivities.
- Reserve imipenem (a potent inducer of beta-lactamases) for proven antibiotic-resistant cases.

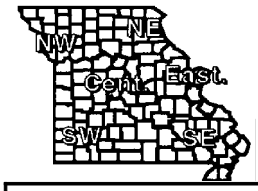
State Public Health Laboratory Report

Newborn Screening — Hypothyroidism, Phenylketonuria, Galactosemia and Hemoglobinopathies

James Baumgartner, B.S., M.B.A., Chief, Metabolic Disease Unit

	Jul 94	Aug 94	Total YTD
Specimens Tested	10,031	11,206	79,858
Initial (percent)	64.3%	64.3%	51,516
Repeat (percent)	35.7%	35.7%	28,342
Specimens: Unsatisfactory	131	142	949
HT Borderline	920	981	6,292
HT Presumptive	46	41	303
PKU Borderline	9	3	72
PKU Presumptive Positive	0	0	2
GAL Borderline	205	188	814
GAL Presumptive Positive	3	2	26
FAS (Sickle cell trait)	102	87	733
FAC (Hb C trait)	22	26	204
FAX (Hb variant)	8	9	88
FS (Sickle cell disease)	0	2	14
FSC (Sickle C disease)	0	0	3
FC (Hb C disease)	0	1	3

HT = Hypothyroidism, PKU = Phenylketonuria, GAL = Galactosemia, Hb = Hemoglobin, YTD = Year to Date



Missouri Deparment of Health
Disease Prevention - Communicable Disease Control
BIMONTHLY MORBIDITY REPORT

Reporting Period *
July - August, 1994

	Districts							KANSAS CITY	ST. LOUIS CITY	sr. LOUIS CO.	SPGFLD GREENE CO.	2 MONTH STATE TOTALS		CUMULATIVE		
	** NW	NE	CD	SE	** SW	** ED	*** OTHER					1994	1993	FOR 1994	FOR 1993	5YR MEDIAN
Vaccine Preventable Dis																
Chickenpox	67	17	28	57	44	38		0	0	0	0	251	171	8093	7463	7463
Diphtheria	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Hib Meningitis	0	0	0	0	0	0		0	0	0	0	0	0	4	5	27
Hib Other Invasive	0	0	0	0	0	0		5	0	1	2	8	12	35	73	
Influenza	0	0	0	0	0	0		0	0	0	0	0	0	158	247	216
Measles	0	0	0	0	0	0		0	0	0	0	0	0	160	1	1
Mulnps	0	1	1	1	0	1		0	1	0	0	s	3	25	25	27
Pertussis	2	0	3	3	1	1		0	0	2	0	12	48	30	80	67
Polio	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Rubella	0	0	0	0	0	0		0	0	0	0	0	0	2	1	1
Tetanus	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
Viral Hepatitis																
A	7	1	4	2	2	17		3	49	34	11	130	165	350	1064	500
B	7	3	1	4	9	8		7	63	11	5	118	85	345	392	392
Non A - Non B	5	0	0	0	1	0		0	0	1	0	7	4	13	21	21
Unspecified	0	0	0	0	0	0		0	0	0	0	0	3	0	13	13
Meningitis																
Aseptic	5	0	5	5	3	6		1	0	9	9	43	80	103	142	137
Meningococcal	2	1	0	1	0	0		0	3	2	0	9	4	43	26	23
Other	0	0	2	1	1	1		1	0	0	0	6	3	42	40	44
Enteric Infections																
Carnpylobacter	16	10	21	14	17	16		12	10	40	14	170	157	437	417	383
Salmonella	25	4	41	21	5	18		24	9	38	8	193	94	409	287	366
Shigella	39	0	0	15	3	1		32	14	19	13	136	170	302	487	312
Typhoid Fever	0	0	0	0	0	0		0	0	0	0	0	0	1	2	2
Parasitic Infections																
Amebiasis	0	0	1	1	0	0		0	0	4	0	6	9	21	29	15
Giardiasis	14	6	7	18	21	20		25	15	27	6	159	130	401	401	428
Sexually Transmitted Dis.																
AIDS	7	1	5	8	4	3	0	27	28	14	7	104	123	501	1417	396
Gonorrhea	49	30	85	106	51	17		407	1140	550		2435	2524	8265	8181	11848
Genital Herpes	33	7	53	27	65	42		121	99	164		611	658	2427	2446	2319
Nongonoc. urethritis	6	9	23	26	6	3		260	688	53	9	1083	1287	4085	4254	4870
Prim. & Sec. syphilis	1	0	0	3	0	0		11	119	51	1	186	252	722	925	340
Tuberculosis																
Extrapulmonary	1	0	1	0	0	0	0	0	0	2	0	4	13	25	26	28
Pulmonary	2	4	4	7	2	2	2	3	0	4	2	32	38	135	145	137
Zoonotic																
Animal Bites	159	65	97	152	126	108		0	1	2	13	723	1146	3365	4413	3878
Psittacosis	0	0	0	0	0	1		0	0	1	0	2	0	4	1	0
Rabies (Animal)	0	0	1	2	0	0		0	0	0	0	3	9	13	19	19
Rocky Mtn. SP. Fever	2	0	1	2	1	0		0	0	0	1	7	9	11	14	18
Tularemia	1	1	1	3	0	1		1	0	0	0	8	5	16	12	28

Low Frequency Diseases

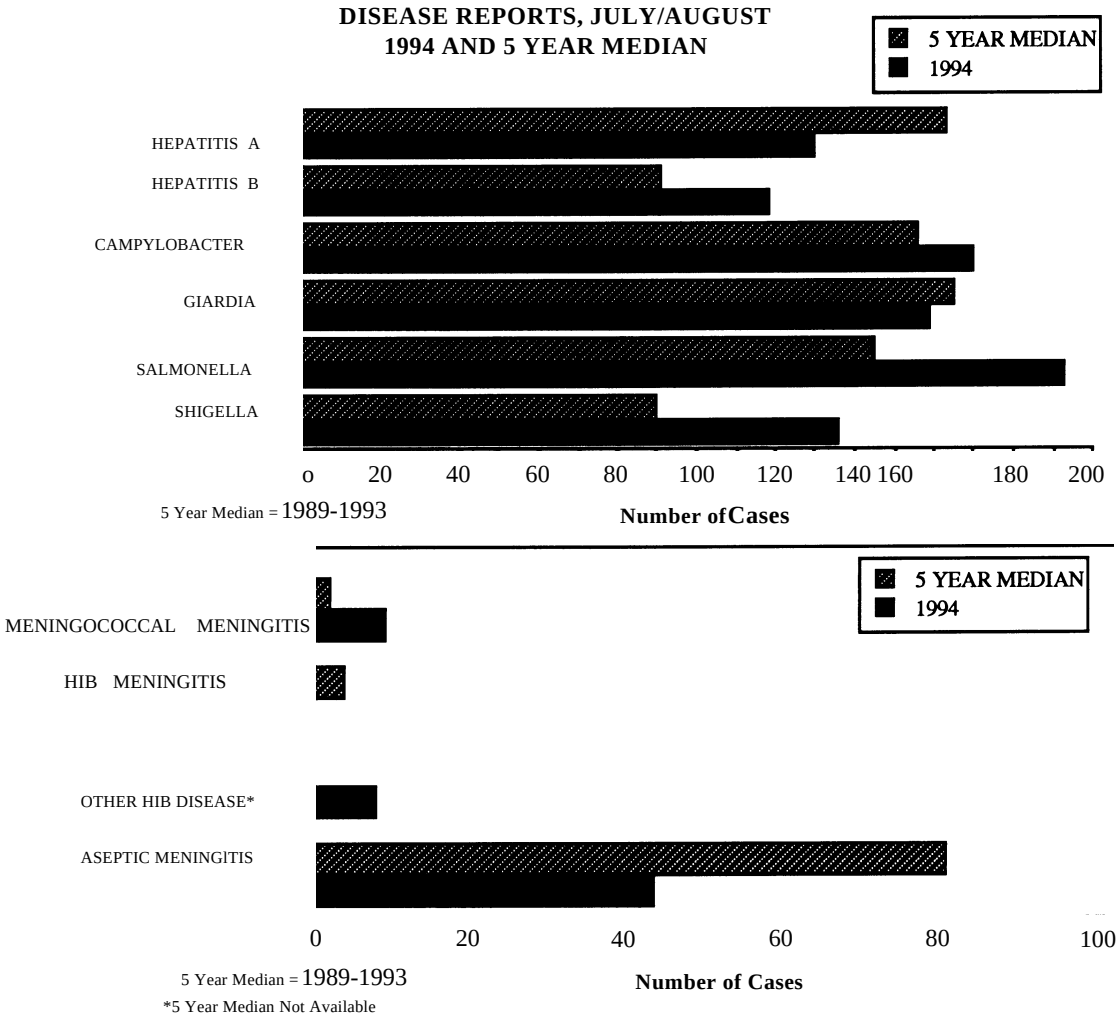
Anthrax
Botulism
Brucellosis
Chancroid
Cholera
Cryptosporidiosis - 8
Encephalitis (infectious)
Encephalitis (viral/arbo-viral)
Granuloma Inguinale
Kawasaki Disease - 1
Legionellosis - 7
Leptospirosis
Lymphogranuloma Venereum
Malaria - 2
Meningococcal, other- 6
Plague
Rabies (human)
Reye Syndrome
Rheumatic fever, acute - 2
Toxic Shock Syndrome - 1
Trichinosis- 1

Outbreaks

Foodborne- 10
Waterborne - 2
Nosocomial - 6
Pediculosis
Scabies- 1
Other
AGI- 1
Meningococcal- 1
Shigella- 1
Hepatitis A - 2

*Reporting Period Beginning July 3, Ending September 3, 1994
**Totals do not include KC, SLC, SLCo, or Springfield
***State and Federal Institutions
** Data not available

Due to data editing, totals may change.



VIRAL HEPATITIS

During the July/August bimonthly period, hepatitis A incidence continued to fall to pre-1992 levels. It decreased by 21.2%, from 165 cases during July/August 1993 to 130 cases during July/August 1994. This is 20.2% below the five year bimonthly median of 163 cases. Hepatitis B cases increased for the period, by 38.8%, from 85 in 1993 to 118 in 1994. This is up 29.6% from the five year bimonthly median of 91 cases.

ENTERICS

Campylobacter rose by 8.3%, from 157 cases during the bimonthly period in 1993 to 170 cases in 1994. This is 9.0% above the five year median of 156 cases. Salmonella at 193 cases, has risen 105.3 to from 94 cases in 1993, and 33.1% from the five year median of 145 cases. Shigellosis decreased 20.0 to from 170 cases in 1993 to 136 cases in 1994. It is up 51.1% from the five year median of 90 cases.

PARASITES

There were 159 giardiasis cases reported in 1994 during the bimonthly period, 22.3% more than the 130 cases reported in 1993. This is 3.6% less than the five year median of 165 cases.

MENINGITIS

Aseptic meningitis decreased by 46.3% to 43 cases in 1994 from 80 cases in 1993. The 1993 bimonthly time period is also the five year median. Meningococcal meningitis increased by 125.0 to to 9 cases in 1994 from 4 cases in 1993. This is a 350.0% increase from the five year median of 2 cases.

HIB DISEASE

There were no cases of Hib meningitis reported for the period in 1994 or 1993, a decrease of 100.0% from the five year median of 4 cases. Other Hib decreased by 33.3%, from 12 cases in 1993 to 8 cases in 1994.

WIND CHILL FACTOR CHART

Cooling Power of Wind on Exposed Flesh Expressed as an Equivalent Temperature (under calm conditions)

Estimated wind speed (in mph)	Actual Thermometer Reading (°F.)											
	50	**40**	**30**	**20**	**10**	**0**	**-10**	**-20**	**-30**	**-40**	**-50**	**-60**
	Equivalent Temperature (°F.)											
calm	50	40	30	20	10	0	-10	-20	-30	-40	-50	-60
5	48	37	27	16	6	-5	-15	-26	-36	-47	-57	-68
10	40	28	16	4	-9	-24	-33	-46	-58	-70	-83	-95
15	36	22	9	-5	-18	-32	-45	-58	-72	-85	-99	-112
20	32	18	4	-10	-25	-39	-53	-67	-82	-96	-110	-124
25	30	16	0	-15	-29	-44	-59	-74	-88	-104	-118	-133
30	28	13	-2	-18	-33	-48	-63	-79	-94	-109	-125	-140
35	27	11	-4	-21	-35	-51	-67	-82	-98	-113	-129	-145
40	26	10	-6	-21	-37	-53	-69	-85	-100	-116	-132	-148
(wind speeds greater than 40 mph have little additional effect.)	**LITTLE DANGER** (For properly clothed person) Maximum danger of false sense of security.				**INCREASING DANGER** Danger from freezing of exposed flesh.			**GREAT DANGER**				

Trenchfoot and immersion foot may occur at any point on this chart.

TEAR OUT FOR FUTURE REFERENCE

INSTRUCTIONS: Measure local temperature and wind speed if possible; if not, **estimate**. Enter table at closest 5°F interval along the top and with appropriate wind speed along left side. Intersection gives approximate equivalent chill temperature—that is, the temperature that would cause the same rate of cooling under calm conditions.

NOTES:

1. Wind may be calm but freezing danger great if person is exposed in moving vehicle, under helicopter rotors, in propeller blast, etc. It is the rate of relative air movement that counts and the cooling effect is the same whether you are moving through the air or it is blowing past you.

2. Effect of wind will be less if person has even slight protection for exposed parts—light gloves on hands, parka hood shielding face, etc.

ACTIVITY: Danger is less if subject is active. A man produces about 100 watts (341 BTUs) of heat standing still but up to 1,000 watts (3,413 BTUs) in vigorous activity like cross-country skiing.

PROPER USE OF CLOTHING and **ADEQUATE DIET** are both important.

COMMON SENSE: There is no substitute for it. The table serves only as a guide to the cooling effect of the wind on bare flesh when the person is first exposed. General body cooling and other factors affect the risk of freezing injury.

Adapted by the Missouri Department of Health from Fort Leonard Wood Form 8-2264 dated June 1991.

Bovine Somatotropin and Milk Safety

Russell Lilly
Bureau of Community Environmental Health

Any time there is change, there will inevitably be people who will oppose it. This has been especially true when it comes to milk. Pasteurization of milk has been common for over 50 years now, and the United States Public Health Service tells us that the risk of contracting disease from drinking raw milk is 50 times greater than if the milk were pasteurized. There are still more than a few who would argue that pasteurization in some way destroys the purity or utility of milk. When milk packaging changed from glass to paper and from paper to plastic, there were those who argued that each change would ruin the product and the industry. Today the concern is about bovine somatotropin (BST).

Recombinant bovine somatotropin (rBST) is a synthetic version of a growth hormone that cattle produce naturally. Posilac, Monsanto's version of rBST, became available to dairy farms in the United States earlier this year. For months preceding and since rBST has been approved for use, there have been individuals and groups who have protested its use, threatened boycotts and tried to convince the public that the product is dangerous both to dairy cattle and to us, the consumers of their milk. These are the very concerns that the Food and Drug Administration addressed before approving the use of the product.

Much of the concern about BST is because it is a hormone. Hormones are chemicals that are produced in one part of the body and have a biologic effect in another part of the body. There are two broad types of hormones: sterodial hormones and protein hormones. Vitamin D is an example of a sterodial hormone; both human and bovine growth hormones are protein hormones. Hormones work by linking up with receptor sites in the part of the body where they are to have their effects. This works much like a key fitting into a lock and is based on the three-dimensional shape of the hormone. All proteins are built from component amino acids, and the shape of the protein is determined by the arrangement of the amino acids. The amino acid sequence of bovine growth hormone is about 35 percent different from that of human growth hormone. Because of this difference, bovine growth hormone has no biologic effect in humans. In the 1950's, before these differences were understood, children with dwarfism caused by a lack of human growth hormone were treated with growth hormone from several species of animals, including bovines. All of these treatments resulted in no benefit or harm to those treated. Even if BST were biologically active in humans, it, like insulin (another protein hormone), would need to be injected to have any effect. When taken orally, protein hormones are broken down into their component amino acids by the enzymes in the digestive tract.

With all of this discussion, you might be thinking that administering additional BST to cattle significantly increases the amount found in milk. This is not the case. When given at prescribed dosages, the amount of BST in the milk remains at a very low level of one to three parts per billion. Only when given about six times the normal dose are milk BST levels substantially increased. When supplementing cattle with rBST, there is a measurable difference in the amount of insulin-like growth factor (IGF-1) in the milk. Normal IGF-1 levels in cow's milk are three to ten ppb and are increased by two to five ppb by treatment with BST. The resulting levels are still no higher than untreated cow's milk in the early stages of lactation. While IGF-1 is normally present in humans and is biologically active, the small increase in the level in treated cow's milk should be of no concern, again, because it is a protein that is digested when taken orally.

One additional safety factor is that most of the BST and IGF-1 in milk is destroyed by pasteurization and about 90 percent is destroyed by the heating processes involved in making infant formula from cow's milk.

Another claim is that the use of BST will cause a large increase in the incidence of mastitis in dairy herds. Mastitis is an inflammation of the udder. This inflammation is caused by infection by various organisms. Supplementing a cow with a growth hormone cannot cause infection or inflammation in the udder. It is a fact that high-producing cattle are naturally slightly more susceptible to mastitis than are low-producing cows. If a cow's production is increased from 50 pounds to 60 pounds per day by any means, e.g., better feed management, improved genetic selection or the use of BST, there will be a slightly higher risk of mastitis. Again, this slightly higher risk is of no concern to the public. When a cow has an infectious organism in her udder, her immune system sends white blood cells to the site to fight the infection. By monitoring the level of somatic cells in milk,we can determine the amount of inflammation and infection. (Milk from uninfected cows still has a level of white blood cells that acts as a defense against infection.) The current legal limit for somatic cells in grade A milk is 750,000 per ml. (down from 1,500,000 per ml. several years ago). In Missouri, our average cell count is normally about 400,000 per ml., which is well below any level of concern. Mastitis is an extremely expensive problem on dairy farms, and if the use of BST significantly increased the level of mastitis, no dairyman would be willing to use it.

A related argument is that if the use of BST increases the level of mastitis, then dairymen will use more antibiotics to treat the mastitis, and there will be more antibiotic residues in the milk. While antibiotic residues in milk is a legitimate issue (because of allergic reactions, development of bacterial resistance, and in the case of some animal drugs, e.g., sulfamethazine, a concern about carci-

nogenic affects), it is not a correct assumption that the use of BST will result in increased levels of residues in consumers' milk. This is true for two reasons. As previously discussed, the actual increase in the level of mastitis should be minimal. Secondly, in the last five years the industry has enacted mandatory programs to reduce the possibility of antibiotic milk reaching the marketplace. This has included producer education and the screening of each truckload of milk for drug residues before the milk is processed. Currently, less than one-tenth of one percent of all loads are destroyed because they contain actionable levels of drug residues.

Some object to BST because it is a product of biotechnology. To manufacture BST, scientists take the part of a cow's genetic information which tells the cow how to produce the protein of BST and inserts it into the genetic code of a harmless bacteria. The bacteria then produces bovine growth hormone. A similar process is involved in producing human insulin. The detractors are concerned that by transferring genes a "super bug" bacteria might be produced. The scientific community is certain that this is not possible.

There are many who say, "At least label the milk and products from cows treated with BST. Don't we have the right to know?" This sounds fine on the surface, but with a deeper look, it is not a good approach. If on a grocer's shelf one jug of milk has nothing on the label and another jug has the statement, "This milk is from cattle treated with BST," many consumers would look at the labeled milk, and because of a lack of information or misinformation, would say, "Oh, that's artificial milk" and not choose it. When, in fact, there is no scientific way to differentiate the milk in the two jugs. If labeling becomes common, the cost to the industry to run two trucks for separate collection and having separate production runs in the processing plants will be tremendous. The likely result would be that all milk will cost more, and all of this is to allay unfounded concerns of the consuming public. If labeling becomes common, there will be processors who will choose not to accept any milk from cows treated with BST. This would deny some producers the ability to use an approved, safe technology that could increase their efficiency and maintain their competitiveness.

With a world population of over five billion and growing every day, we cannot afford to ignore and turn our backs on new food producing technologies. The companies involved in producing commercial BST have invested millions of dollars, and if the use of this product is limited by outside constraints, will companies be willing to invest in future food producing technology?

The good news is that, like pasteurization and plastic milk jugs, BST appears to be accepted in the industry. Since its introduction, over 10,000 dairy farms have used the product, and national figures show no decline in milk consumption.

Early Syphilis in Bootheel Region

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performed for all patients who may be at risk for the disease. In addition, HIV has been determined to be more easily transmitted during the presence of syphilitic lesions. It is, therefore, important to counsel and test those patients at risk for syphilis for HIV as well.

Increases in the annual number of reported HIV cases have been observed in recent years in the seven-county Bootheel area. In 1991, seven cases of HIV were reported followed by 13 cases (an 86% increase) in 1992. Seventeen HIV cases were reported in 1993, a 31 percent increase over the previous year. Based on the first nine months of 1994, it is projected that 32 cases of HIV will be reported from the Bootheel area over the course of the year, which would represent an 88 percent increase over cases reported in 1993. See Figure 4.

All cases of syphilis, HIV and AIDS, as well as other communicable diseases, are reportable by statute and/or regulation in Missouri. It is critical that all cases of diagnosed or suspected syphilis and/or HIV be promptly reported in order to initiate the disease intervention process as rapidly as possible. Cases must be reported to the local health department, the district health office or the Bureau of STD/HIV Prevention in Jefferson City. Disease intervention follow-up by public health personnel will occur only after contact with the patient's attending physician.

Report cases to:

Bureau of STD/HIV Prevention
P.O. Box 570
Jefferson City, MO 65102
Telephone: (314) 751-6139
Secure Fax: (314) 751-6417

Big River Lead Exposure Study

(continued from page 5)
extent of lead poisoning in children in the United States: a report to congress. Agency for Toxic Substances and Disease Registry, 1988.
29. Perino J, Ernhart CB, The relation of subclinical lead level to cognitive and sensorimotor inpairment in black preschoolers. J Learn Disabil 1984;7:616–20.
30. Sullivan LW. Keynote address presented at International Meeting, Alliance to End Childhood Lead Poisoning, Washington,DC; October 7, 1991.
31. HUD (U.S. Department of Housing and Urban Development). Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing. Report to Congress, Washington DC; HUD, 1990.

Guidelines for Scabies Prevention and Control

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Introduction

Since 1989, approximately 90 clusters or outbreaks of scabies have been reported to the Bureau of Communicable Disease Control. The majority of these reports have come from long-term care facilities (LTCFs), although there are occasional reports from hospitals, day care centers and schools. Requests for assistance in resolving outbreaks in some LTCFs have uncovered probable scabies infestations lasting a year or longer. We have had reports of symptoms developing ten days following exposure; however, most cases have an incubation period of four to six weeks for a primary infestation.

A long incubation period (during which time the mite [*Sarcoptes scabiei* var. *hominis*] is able to be transmitted to close contacts) and a wide variety of presentations are problematic in getting an accurate diagnosis. Because scabies can present with burrows, papules, scales, vesicles, bullae, crusts, pustules, nodules and excoriations, it is necessary to do a careful history followed by burrow identification and skin scrapings for the mite, its eggs or fecal pellets. The following are recommendations for prevention and control of institutional scabies.

Scabies Prevention Programs in Health Care Facilities Require That:[1]

1. Health care workers be suspicious of scabies in persons with a rash or pruritus that has gradually gotten worse, particularly during the night time hours;

2. Health care facilities establish a policy of examining newly admitted persons for scabies and questioning new employees for either exposure to or symptoms of scabies;

3. The diagnostic skills of a consultant experienced in recognizing scabies be used in evaluating difficult or unusual cases;

4. In-house competence in preparing and examining skin scrapings from suspect persons be developed;

5. Protective clothing and gloves be used when providing hands-on care to persons suspected of having scabies;

6. A system for recording epidemiologic and clinical information on suspect and confirmed persons be established.

Equipment Needed for Skin Scrapings:

1. Gloves
2. Magnifying glass
3. Gooseneck lamp
4. Felt tip pen—green or blue washable ink
5. Alcohol swabs
6. #15 scalpel blades, glass slides for scraping, or curettes
7. Scalpel holder
8. Kelly clamp or other forceps
9. Mineral oil
10. Slides and cover slips
11. Requisitions, if slides are being sent to a public health laboratory
12. Sharps container
13. Clear nail polish
14. Microscope

Procedure for Doing Skin Scrapings[1,2]

1. Establish and confirm the diagnosis by skin scrapings and microscopic identification of mites, eggs or scybala (fecal pellets). A nurse from the facility can be taught this procedure by a dermatologist, the consulting physician or by a nurse or technician who has had professional training in doing the procedure.

a. Mass treatment should not be initiated unless a definite diagnosis has been made in at least 1 of the symptomatic cases.[1]
b. Scrape those persons with the most severe rash first. Elderly may present with severe urticaria and bullous lesions.
c. Shoulders, back and abdomen are choice areas for scrapings in the elderly.[2] Other sites: hands, wrists, elbows, feet, ankles, buttocks, axillae, knees, thighs and breasts.
d. Use hand magnifying lens to identify recent burrows or papules. A bright light and magnifying glass will assist in visualizing the tiny dark speck (the mite) at the end of the burrow.
e. Identify these high yield lesions by applying mineral oil (best used over dry scaly areas) or by applying the burrow ink test to possible burrows. The burrow ink test is done by using a wide felt tip pen (blue or green are best) over burrows and then wiping off with an alcohol swab. The alcohol will remove most surface ink, but will not remove the ink taken up by the burrow, thus leaving a dark irregular line.
f. Apply mineral oil or preferably microscope immersion oil to lesions or scalpel blade and glass slides.[2]
g. Scrape non-excoriated, non-inflamed areas (burrows and papules) vigorously with a #15 scalpel blade or glass slide held at a 90º angle to the skin and while holding the skin taut until the stratum corneum is removed.[2,3,4] (Vigorous scraping appropriately results in a few red blood cells visible under the microscope, but there should not be frank bleeding.) Some practitioners prefer using a small curette. Change blades or curettes between scrapings on different persons. Blades can be placed and removed from the handle with a forceps. Used blades must be placed in a sharps container.
h. Transfer skin scrapings from 6 different sites to a single slide or to 6 different slides per patient.[2] These scrapings can be pushed onto the slide

Table 1. Definitions of Scabies Infestations

Conventional scabies: average 10-15 mites at any given time, although only 1–2 mites may be recovered in scrapings, (frequently none are observed); occurs in physically healthy persons.[1,2]

Severe scabies: **Atypical** crusted scabies: usually a total of 3–6 mites and 8–12 eggs observed on 5–7 slides; do not exhibit hyperkeratotic cutaneous response because of decreased cell mediated immunity; some lack pruritus; occurs in nursing home residents and elderly with coexistent chronic disease; moderate to high risk of transmission.[6]

Norwegian scabies: **Typical** crusted or keratotic: thousands of mites at any given time; multiple live mites, eggs, and scybala (fecal pellets) observed on almost every slide; have hyperkeratotic skin; occurs in debilitated, immunosuppressed, advanced chronic disease and mentally handicapped. Risk of transmission is high from skin and fomite contact. (Exfoliating skin scales harbor enormous numbers of mites which are shed onto linens, furniture, and carpeting).[1,2,5,7]

Nodular scabies: pruritic nodules, apparently due to hypersensitivity persisting for weeks to a year or longer, despite scabicidal therapy, but eventually clear spontaneously; may regress with use of corticosteroids; surgical excision sometimes indicated if patient concerned and intralesional corticosteroids ineffective.[5]

Pseudoscabies: scrapings always negative; fostered by residual pruritus in effectively treated cases and by conversations between misinformed persons.[1-5]

Canine-transmitted scabies: caused by the *Sarcoptes scabiei* var *canis* species of mite from dogs; the mite does not reproduce or complete its life cycle on humans and thus burrows are not created; not usually transmitted person to person; as a rule self-limiting in humans.

edge and then moved to the center of the slide.

i. Place a cover slip over the slide.[2]

j. Examine entire slide methodically under low power at 25–50 x magnification for at least 5 minutes.[1] Low power (2.5–4 x) is useful initially. The microscope should be taken to the facility; however, if the practitioner is not trained in reading the slides, the cover slip should be secured to the slide at all edges with clear nail polish and transported personally, by courier, or by mail (in a secure mailer) to:
   1) Missouri State Public Health Laboratory (MSPHL);
   2) a branch of MSPHL;
   3) a hospital or rural clinic laboratory with pre-arrangements; or
   4) a physician's office with pre-arrangements.

   Public health laboratory requisitions must accompany slides if readings done at public health laboratories.

Epidemiologic Variables for Scabies[1,2]

1. Make a line list* of room number, age, sex, symptoms, date of onset for:

a. **Symptomatic persons with positive scrapings;** differentiate between conventional and Norwegian (keratotic or crusted) scabies.[1,2,5] See Table 1 for Definitions of Scabies Infestations.
b. **Symptomatic persons with negative scrapings**
c. **Asymptomatic contacts** of a symptomatic case. These contacts should be on a totally separate line list. Close contacts are persons who have skin to skin contact, sleep in the same bed or handle infested clothes and bed linens. Contacts of crusted scabies should be designated High Risk, Low Risk and No Risk per definitions on page 18.
d. Contact tracing should go back 2 months.

2. Ascertain the epidemic level: proportion of affected persons (positive scrapings or symptomatic).[1] This information will determine whether persons in the whole facility or just one section are treated.

a. Determine percentage of affected persons (patients or residents) within the entire facility's population of patients or residents.
b. Determine percentage of affected employees within the entire facility's employee population. Use questionnaire provided by DOH.*
c. Determine percentage of affected persons within each subgroup of a population; i.e., nursing home wing, hospital department.

3. Look for similarities or groupings in age and sex among affected persons.[1]

4. Ascertain type and frequency of secondary bacterial infections.[1,5]

5. Determine the mode of transmission; i.e., employees having close personal contact like bathing, bedmaking, applying skin lotions, frequent lifting/repositioning of patients[1,2]

or

exchanging clothing, sleeping on same linens, playing games involving close hand or skin contact[1,2]

or

sexual contact.[1,2]

General Recommendations

1. Report outbreak to the local health department using an outbreak report form.* Do not use separate CD-1 cards for every case in an outbreak.

2. Notify facilities to which potentially infested patients or employees have transferred.[1,8]

3. Intensive educational programs should be given to all employees.[1] They should be given a Fact Sheet on Scabies.

4. Allocate sufficient personnel and funding to initiate and manage follow up treatments. Facility should purchase

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(continued from page 15)
enough medication to treat symptomatic persons (patients/residents, employees, volunteers and family members) and their close contacts.[1,2]

Selective Treatment Protocol[1]

1. A conventional scabies treatment regimen can be selective when 1 person has a positive scraping (which is **not** indicative of Norwegian scabies), See Table 1 on page 15 for Definitions of Scabies Infestations. Selective treatment protocol can be used.[1]

2. The diagnosed and probable infested **cases** and **symptomatic contacts** should receive treatment with subsequent monitoring for effectiveness of treatment. A skin scraping should be done on the symptomatic cases 1 month after treatment,[2] particularly if rash and symptoms persist. See section on Application of Scabicides starting on this page.

3. All "hands-on" **contacts** during preceding 2 months (employees, relatives and other patients) of this patient/resident and close personal **contacts** of the symptomatic employee should receive treatment.[1,2,9]

Mass Treatment Protocol[1,2,9]

1. A physician should be designated as the outbreak control officer and be given authority to manage the treatment regimen of all residents in a long term care facility. At the least, all attending physicians should agree to a cooperative schedule for conventional or Norwegian scabies.[9] See Table 1 on page 15 for Definitions of Scabies Infestations.

2. Mass treatment should be administered within a 24–48 hour period to all persons (residing and working) in a defined area of the facility if:[2,9]

• 2 or more symptomatic patients/residents have positive scrapings and 1 or more employees on the same unit exhibit pruritus or have a positive scraping[2]

or

• 1 asymptomatic patient/resident has a positive scraping and many patients/residents have exhibited symptoms of infestation for months (2–10% rate of symptomatic infestation).

or

• Norwegian scabies is diagnosed in 1 patient/resident and at least 1 employee is symptomatic.[1]

3. Mass treatment of everyone in the facility (all residents, at risk employees and household members) should be administered within a few successive days if positive scrapings are found in 2 or more separate areas of the facility.

4. Employee crossover should not be allowed until the specified population has been treated.

5. Household members, sexual contacts and roommates of symptomatic employees should be treated the same day as the employees.[1]

6. Write a detailed schedule of:

a. Who will be treated and who will do the treating;
b. What will be used for treatment, including specific instructions on how to apply lotions;
c. Where treatments will be done; i.e., a treatment room, individual beds, at home;
d. When treatments will be done (date and time);
e. State when the person will be considered non-infested, can be removed from isolation and can return to work. See section on Isolation and Environmental Control for Conventional Scabies on page 18.

7. Write a second schedule for:

a. Reassessment of all treated persons at 14 days.
b. Persons needing a second treatment 3–7 days later. See subsections 8 and 9 under Application of Scabicides on pages 17–18.
c. Persons with crusted or infected lesions needing routine daily monitoring, monthly scrapings for a few months or a maintenance monthly treatment regimen.[2]

8. Notify all families and frequent visitors about problems and need for their cooperation.[1,2]

Application of Scabicides and Steroid Creams

1. Treatment failures may occur for several reasons, the most common being **inadequate application of scabicide**.[1,2,5,8–10] Other reasons for treatment failure include:

a. infected or crusted lesions.[9]
   1) Keratolytic agents (20–40% urea and 6% salicylic acid) may be necessary to soften scaliness and permit penetration of scabicide.[2,5,11]
   2) Concomitant bacterial infection should be treated with appropriate antibiotics and retreated for scabies a week or 10 days later.[11]
b. reinfestation from untreated contacts [10]
c. cell-mediated immunodeficiency [1,12]
d. resistance of mites to the scabicide [8,13,14]

   NOTE: Pruritus and rash can continue for 1–4 weeks after treatment. Pruritus and residual rash should not be considered treatment failure until 1 month after last treatment. To ameliorate these signs and symptoms, some dermatologists use 1% hydrocortisone cream or triamcinolone cream (0.1%–0.025%) applied to the most intense rash and a lubricating agent or emollient to the lesser rash for children,[15,16] 1% hydrocortisone cream or triamcinolone cream 0.1% can be used for adults as well.[15] Antihistamines are also used to alleviate the hypersensitivity response.
e. Steroid creams should not be applied until after first scabicide treatment. Topical and systemic steroids cause depression of delayed hypersensitivity and pruritus, thus allowing scabies to go undetected and transmission unimpeded.

2. Gloves and gown are worn to apply scabicides.

3. Bathe as usual and change bed linens.

4. Apply scabicide to every square inch of skin, from the posterior ear folds down over entire body, including all non-affected areas. Include intergluteal cleft, navel, crevices of contractured extremities, and webs between fingers and toes.[11] If scabicide is washed off during handwashing or perineal care, it must be reapplied.

5. In infants and young toddlers, the elderly and the immunocompromised, the head (face and scalp) requires application of scabicide. Pay close attention to the area behind the ears. Do not get the scabicide near the eyes or mouth. Prior treatment failure may be an indication to include the head in other persons.[2,11]

Lindane shampoo, used as directed on the label, can be used for certain persons (elderly) to treat the scalp.

6. Fingernails and toenails should be clipped and scabicide applied under nails. A small soft brush is helpful for this.[2,17,18]

7. Scabicides

a. 5% permethrin cream (a synthetic pyrethroid)[19]
   - Considered drug of choice by several authorities including the 1994 American Academy of Pediatrics "Red Book" and The Medical Letter, March 23, 1990, p. 29.
   - Cure rate in one study was 91%.[10,14]
   - 1 application is considered curative, although 2 applications are frequently recommended by experts for symptomatic persons.

The usual adult dose is 30 grams. A 60-gram tube should treat 2 adults. For adults, it should be massaged into the skin covering the entire body (except the head) from the soles of the feet to the neck. For infants, young toddlers, and geriatric patients, it should be applied to the entire body including the scalp, neck, temples and forehead because the mite often infests these areas in those age groups. The patient should be instructed to remove the medication by thoroughly bathing 8–14 hours after application. Contact with the eyes and mouth should be avoided. If contact occurs, the eyes should be immediately flushed with water. Note: Studies have not demonstrated plasma levels. The drug is rapidly broken down and is excreted in urine as inactive metabolites.[6,19]

Permethrin is safe for children 2 months of age and older. No instance of accidental ingestion has been reported. The most commonly reported side effects are pruritus, edema and erythema, which may continue for up to 2 weeks after treatment. Patients should be told that the itching or stinging of scabies infestation may continue after treatment, and should be advised to avoid repeated application of the scabicide.

Although animal studies showed no adverse effects to reproductive function or damage to the fetus, no adequate studies have been done on pregnant women. Therefore, permethrin should be used during pregnancy only when clearly necessary. If treatment is necessary for lactating mothers, breast-feeding should be discontinued during the treatment period.

b. 1% lindane cream or lotion is effective when applied **properly**.[9,11,20] The usual amount of lindane lotion required to treat one adult once is 30 grams (1 oz.).[6] Lotion bottle must be **shaken well**.
   - Leave on for 8 hours or overnight; some physicians prefer a 12–24 hour application.[5] Most absorption of lindane occurs in the first 6 hours after application.[20]
   - Avoid contact with eyes and mucous membranes.
   - Not to be used for small infants, pregnant women or nursing mothers.[10,20] Use of lindane for any reason in small children is seriously questioned by the National Pediculosis Association. Lindane should be avoided in anyone with seizure disorders and in anyone with severe skin disruption (excoriated or denuded). If lindane is used for a lactating mother, discontinue breast-feeding for 2 days.[6]

c. 6% precipitated sulfur in petrolatum prepared by pharmacy.[15]
   - Cure rate is unknown—has not been studied, but used for centuries.
   - Product is messy, malodorous and somewhat irritating.
   - Apply nightly for 3 nights (wash off previous application before reapplying a new application).[15]
   - Recommended in infants younger than 2 months of age and in pregnant or lactating women.[15]

d. 10% crotamiton cream or lotion has an approximate 50% cure rate when applied less than 5 days, [10,20–22] 60% effective for full treatment.
   - Cream must be thoroughly massaged into skin.
   - Apply twice a day for 5 days.[10]
   - Avoid contact with eyes and mucous membranes.
   - Can be used on youngsters and elderly with dry sensitive skin,[5] but not denuded skin.[20]

8. Conventional scabies regimen

a. A single application of 5% permethrin cream or 1% lindane is recommended in facilities provided that application of scabicide is supervised by a professional health care worker who is knowledgeable about scabicide treatments. Several authorities claim that a single adequate application of 5% permethrin cream or 1% lindane is sufficient to eradicate conventional scabies, whether a diagnosed case, symptomatic case, or asymptomatic contact.[9,11] This has been effective in the clinical practice of treating individual families.

b. Institutional scabies has a high propensity for transmission. If supervised application of scabicide by trained employees is not possible, the following regimen is recommended:

   Persons who are positively diagnosed by skin scrapings—
   - 3 treatments spaced 3–7 days apart, utilizing 2 different agents[2]
   - reevaluate at 14 and 28 days.

   Symptomatic cases whose skin was not scraped or scraping was negative—
   - 2 treatments, 3–7 days apart [2,5,11]
   - reevaluate at 14 and 28 days.

(continued on page 18)

(continued from page 17)

Asymptomatic contacts, including household and sexual contacts, of diagnosed or symptomatic cases—
- 1 treatment, evaluate in 14 days[2]

c. It should be acknowledged that some clinicians prefer to treat symptomatic individuals with two applications on two consecutive days.

9. Norwegian scabies (atypical, crusted) regimen

a. Aggressive treatment over entire body.[1] See subsections 1–6 under Application of Scabicides on pages 16–17.
b. 5% permethrin cream for 1 day, followed by 10% crotamiton lotion for 5 days, followed by a second 5% permethrin cream for 1 day.[2,5,8]
c. Reassess on days 7 through 14 with follow-up scrapings in one month.[2] If scrapings are positive or if symptoms unabated, treat again.
d. If treatment failure occurs several times, monthly maintenance treatments should be given for an extended period of time; (e.g., applications of 10% crotamiton lotion for 2 days each month.[2,8]
e. Protective gown and gloves are necessary until scrapings are negative on 3 separate occasions.
f. Categorize contacts by risk of mite transmission[1]

1) High risk: prolonged or recurrent hands-on contact before initiation of patient treatment,
- 2 treatments, 3-7 days apart.

2) Low risk: persons having had indirect contact (touching patient's clothing or linens); a simple, brief period of direct skin to skin contact (obtaining a blood specimen, positioning a patient for radiography); or a patient who was cared for by an employee who also cared for the scabietic patient.
- 1 treatment

3) No risk: persons having had neither direct nor indirect contact require no treatment.

10. Cleansing bath is taken when product is to be removed. Some experts do not believe it is necessary to bathe residents at designated times in order to remove scabicide. Estes and Estes suggest that an extended interval before bathing or repeated applications be considered to offset reinfestation.[6]

11. Fresh clean linens and clothes are put on after the cleansing bath.

Isolation and Environmental Control for Conventional Scabies

1. Environmental reservoirs were considered to play little or no role in scabies transmission until late 1988. Since then, Arlian and colleagues have demonstrated that *S. scabiei* can remain alive for 3 days on stuffed chairs, sofas and tiled floors. He found that nymphs could survive 2–5 days at 25°C and 45–75% relative humidity. Outbreak reports implicate laundry and clothes as probable sources of transmission.[23]

2. Isolate affected patients/residents during the treatment period or for 24 hours after initiation of scabicide such as 5% permethrin cream or 1% lindane lotion; 24 hours after last application of other scabicides; restriction of contact with others—restrict to room or home.[5]

3. Wear gown and gloves for skin to skin contact. Wash hands after removal of gloves.[1]

4. Bed linens, towels and clothes used by the affected persons within 72 hours prior to treatment should be placed in plastic bags inside the patient's room, handled by gloved and gowned laundry workers and laundered at 50° C (122° F).[1,23,24] Hot cycle of dryer should be used for at least 10–20 minutes. Nonwashable blankets and articles can be placed in a plastic bag for 7 days, dry cleaned or tumbled in a hot dryer for 20 minutes.[26]

5. All bed linens, towels and clothes should be changed daily.

6. Multiple-use walking belts, skin creams and ointments can serve as potential reservoirs for mites. Disinfect the walking belt and discard all creams, lotions or ointments used prior to effective treatment.[25,26]

7. Mattresses, upholstered furniture and carpeting should be vacuumed.

8. Routine disinfection procedures are adequate on a daily basis.[1]

9. Symptomatic employees should be allowed back to work the morning following overnight treatment with 5% permethrin cream or 1% lindane.[1] Disposable gloves should be worn for 2–3 days by symptomatic staff who must provide extensive hands-on care to their patients.[1]

Isolation and Environmental Control for Norwegian Scabies -
(Measures remain in place until skin scrapings are negative on 3 consecutive occasions.)

1. Assign patient/resident to a private room.[1]

2. Restrict contact with visitors until treatment regimen completed and scrapings are negative for live mites. Alternatively, visitors must take the same precautions (wearing a gown and gloves) as employees.[1,2,27]

3. Cohort employees to care for this patient/resident only (no other direct care responsibilities) until effective treatment is completed. Other duties for these employees can include record keeping and filing.[1]

4. Wear gown and gloves to attend to patient needs, for housekeeping duties and handling of laundry.[28]

5. Spray insect repellent (pyrethrins) to wrist (edge of glove and ribbing of sleeve area), arms and front of gown. Remove before leaving the room. Wash hands.

6. Upholstered furniture covered with cloth fabric should be removed from the room or replaced with furniture covered in plastic or vinyl. Mattresses must be covered with plastic or vinyl.[1]

7. The patient's room should be vacuumed daily with a vacuum cleaner designated for this room alone.[1]

8. Routine disinfection procedures should follow thorough vacuuming on a

daily basis and upon discharge of the patient from the room.

9. Utilize any other appropriate protocols such as given in subsections 4–6 under Environmental Control for Conventional Scabies on page 18.

REFERENCES

1. Juranek DD, Currier RW, Millikan LE. Scabies control in institutions. In: Orkin M, Maibach HI, (eds.) Cutaneous Infestations and Insect Bites. New York: Dekker, 1985: 139-156.
2. Currier RW. Scabies and pediculosis: hospitalized mites and lice. Asepsis - The Infection Control Forum 1984; 6:13-21.
3. Muller GH. Laboratory diagnosis of scabies. In Orkin M, Maibach H, Parish LC, Schwartzman RM (eds): Scabies and Pediculosis. Philadelphia: Lippencott, 1977.
4. Muller G, Jacobs PH, Moore NE. Scraping for human scabies - a better method for positive preparations. Arch Dermatol 1973; 107:70.
5. Orkin M, Epstein E, Maibach HI. Treatment of today's scabies and pediculosis. JAMA 1976; 236:1136-1139.
6. Estes, A, Estes J. Therapy of Scabies: Nursing Homes, Hospitals, and the Homeless. In: Maibach HI. Seminars in Dermatology 1993; 12 (March): 26-33.
7. Carslaw RW, Dobson RM, Mood AJK, et al. Mites in the environment of cases of Norwegian scabies. Br J Dermatol 1975; 92:333.
8. Centers for Disease Control. Scabies in health-care facilities - Iowa. Morbidity and Mortality Weekly Report 1988; 37:178-179.
9. Taplin D, Arrue C, Walker JG, Roth WI, Rivera A. Eradication of scabies with a single treatment schedule. J Am Acad Dermatol 1983; 9:546-550.
10. Reeves JRT. Head lice and scabies in children. Pediatr Infect Dis J 1987; 6:598-602.
11. Taplin D. Resistance to antiscabietic drugs. J Am Acad Dermatol 1983; 8:122-123. (Reply)
12. Moberg SAW, Lowhagen GE, Hersle KS. An epidemic of scabies with unusual features and treatment resistance in a nursing home. J Am Acad Dermatol 1984; 11:242.
13. Hernandez-Perez E. Resistance to antiscabietic drugs. J Am Acad Dermatol 1983; 8:121-122. (Letter to Editor)
14. Taplin D, Meinking TL, Porcelain SL, Castillero PM, Chen JA. Permethrin 5% dermal cream: a new treatment for scabies. J Am Acad Dermatol 1986; 15:995-1001.
15. Orkin M, Maibach HI. Scabies Therapy-1993. IN: Maibach HI. Seminars in Dermatology 1993;12 (March):22–25.
16. Rasmussen JE. Scabies. Pediatrics in Review 1994;15:110–113.
17. Witkowski JA, Parish LC. Scabies; Subungual areas harbor mites. JAMA 1984; 252:1318-1319.
18. Scher RK. Subungal scabies. Am J Dermatol 1983; 5:187.
19. Taplin D, Meinking TL. Pyrethrins and pyrethroids in dermatology. Arch Dermatol 1990;126(Feb):213–221.
20. Shacter B. Treatment of scabies and pediculosis with lindane preparations: an evaluation. J Am Acad Dermatol 1981; 5:517-527.
21. Cubela V, Yawalker SJ. Clinical experience with crotamiton cream and lotion in treatment of infants with scabies. Br J Clin Pract 1978; 32:229-231.
22. Kostantinov D, Stanoeva L, Yawalker SJ. Crotamiton cream and lotion in the treatment of infants and young children with scabies. J Int Med Res 1979; 7:443-448.
23. Arlian LG, Runyan RA, Achar S, Estes SA. Survival and infectivity of *Sarcoptes scabei* var. *canis* and var. *hominis*. J Am Acad Dermatol 1984; 11:210-215.
24. Cooper CL, Jackson MM. Outbreak of scabies in a small community hospital. Am J Infect Control 1986; 14:173-179.
25. Burkhart CG. Scabies: an epidemiologic reassessment. Ann Intern Med 1983;89:498–503.
26. Degelau J. Scabies in long-term care facilities. Infect Control Hosp Epidemiol 1992;13:421–425.
27. Clark J, Friesen DL, Williams WA. Management of an outbreak of Norwegian scabies. Am J Infect Control 1992;20:217–220.
28. Thomas MC, Giedinghagen DH, Hoff, GL. Brief report:An outbreak of scabies among employees in a hospital associated commercial laundry. Infect Control 1987;8:427–429

Additional References

- N.Y. State Department of Health. Scabies outbreaks in health care facilities May 3, 1983; Series 83-44. (Memorandum)
- Lettau LA. Nosocomial transmission and infection control aspects of parasitic and ectoparasitic diseases Part III. Ectoparasites. Infect Control Hosp Epidemiol 1991; 12:179-185.
- Taplin D et al. Community control of scabies: a model based on use of permethrin cream. Lancet 1991; 337:1016-1018.
- Taplin D, Meinking TL. Infestations. In Schachner LA, Hansen RC (eds): Pediatric Dermatology, Vol. 2, pp 1487-1501.
- Estes SA. The diagnosis and management of scabies (Monagraph) Reed & Carnick, Piscataway, NJ, Nov. 1988.
- Kolar KA, Rapini RP. Crusted (Norwegian) scabies. American Family Physician 1991;44(4)…1317–1321.
- O'Donnell BF, O'Laughlin S, Powell FC. Management of crusted scabies. Int J Dermatol 1990;29:258–266.
- Lerche NW, Currier RW, Juranek DD et al. Atypical crusted "Norwegian" scabies: Report of nosocomial transmission in a community hospital and an approach to control. Cutis 1983;31:637–642+.

*Additional information and data collection forms for patients, residents or employees are available from the Department of Health (DOH) by contacting the **Bureau of Communicable Disease Control at (314) 751-6115.**

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Mark Your Calendar For Tuberculosis Awareness Fortnight

Each year the American Lung Associations of Eastern and Western Missouri, along with the Missouri Department of Health, Bureau of Tuberculosis Control, co-sponsor Tuberculosis Awareness Fortnight. This up-coming event is scheduled to take place March 12–25, 1995.

All physicians and health care providers are encouraged to participate by providing displays, educational materials and lectures to their staff and clients on the importance of tuberculosis screening, prevention and treatment.

The Bureau of Tuberculosis Control will be sponsoring programs and providing material on tuberculosis control to health care providers and health care workers along with the general public. Several lectures are being planned throughout the state.

The American Lung Association of Eastern Missouri has announced that a Comprehensive Lung Health Conference will be held on Thursday, March 23,1995, at the May Community Education Center at DePaul Health Center in St. Louis. The conference is designed to provide physicians, nurses and other health care professionals with the opportunity to increase their knowledge and understanding of current concepts in the diagnosis, management and prevention of lung disease, with special emphasis on tuberculosis. Among the prominent speakers will be Dr. Joseph Bates, President of the American Lung Association; Dr. Margaret Smith, Professor of Pediatric Medicine at Tulane University; Dr. Samuel Dooley from the Centers for Disease Control and Prevention; and Dr. George DiFerdinando, Director of the New York State Tuberculosis Control Program. CME, CEU and CRCE credits will be offered for the one-day conference.

The American Lung Association of Western Missouri will be sponsoring several presentations by Dr. Charles Peloquin from the National Jewish Center for Immunology and Respiratory Medicine, Denver, Colorado. He will be speaking on "Strategies for Managing Cases of Drug-Susceptible and Multiple Drug-Resistant *M. tuberculosis*," for a Pulmonary Roundtable of Physicians group on Thursday evening, March 23,1995. Two Grand Rounds covering the same topic are planned for Dr. Peloquin on Friday, March 24,1995. One is scheduled at St. Luke's Hospital in Kansas City in the morning, and the other will be held at University of Missouri-Kansas City School of Medicine in the afternoon.

For further information regarding these and other events, or to obtain additional information and literature on tuberculosis contact:

American Lung Associations
of Eastern and Western Missouri
(800) LUNG-USA
or
Bureau of Tuberculosis Control
(314) 751-6122.